# NATURE AND NURTURE ON PICKY EATING BEHAVIOR IN EARLY CHILDHOOD

BY

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DISSERTATION

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## ABSTRACT

Eating behavior in children is influenced by both genetic and environmental determinants. In the first two years of life, children develop food preferences and dietary habits as they transition from a milk-based diet to table foods. During this time, picky eating behavior often emerges as children become more independent during mealtime. Picky eating, or fussy eating, is typically characterized as having a low dietary variety and unwillingness to eat either familiar or novel foods to the extent of causing problematic interference during mealtimes. Previous literature on picky eating has largely focused on school-aged children. In addition, no study had investigated the joint influence of nature and nurture on children's picky eating behavior using a transdisciplinary gene-environment approach. Research is needed to gain a better understanding of the early-life contributors of picky eating behavior and factors that promote healthy dietary patterns, growth, and development in early childhood.

The purpose of this dissertation was to determine the influence of genetics (nature) and the feeding environment (nurture) on picky eating behavior in children aged 24 months from the STRONG Kids 2 longitudinal birth cohort study. Towards this goal, there were three specific aims: 1) identify the association between individual genetic variations related to appetite and picky eating behavior; 2) identify the association between the reported and observed parental feeding responsiveness with picky eating behavior; and 3) determine the interaction between cumulative genetic risk and parental feeding responsiveness in the prediction of children's picky eating behavior. DNA was isolated from saliva samples collected at 6 weeks of age. Mothers completed surveys when the child was 18 and 24 months of age regarding their feeding practices and their child's eating behaviors. Families were observed during a typical family dinner in the naturalistic home setting and videos were coded for parents' observed feeding practices.

Two studies were conducted to investigate the role of nature on picky eating. We identified the association between picky eating and single nucleotide polymorphisms (SNPs) in genes related to chemosensory perception and appetite control. Individual genetic variation in genes related to bitter taste was associated with picky eating, suggesting that children who are sensitive to bitter taste are more likely to be picky eaters. We also identified an association between picky eating and individual genetic variation in genes related appetite regulation, suggesting that picky eaters may have reduced appetite based on genetic differences.

To identify the role nurture on picky eating, we conducted two studies to examine the influence of the feeding environment on picky eating behavior. There was a prospective relation between picky eating and factors related to the home feeding environment. Among preschool-aged children, avoiding the television, maintaining parent control of food choices during mealtimes, and having a higher sense of positive climate during family meals was associated with lower picky eating behavior one year later. Among toddlers, reported parent nonresponsive feeding practices was positively associated with picky eating behavior, suggesting that responsive feeding practices may promote healthful eating behavior in young children.

To investigate the joint influence of nature and nurture on picky eating, we utilized a gene-environment interaction approach based on the differential susceptibility hypothesis. A cumulative genetic risk score was calculated based on genes previously identified in the first study. We found that children with the highest cumulative genetic risk were pickier when they were exposed to an environment with high parental feeding responsiveness, but less picky in an environment with low feeding responsiveness. Overall, these results indicate that both nature and nurture influence the development of picky eating in children and findings can be used to inform the development of anticipatory guidance for parents and caregivers of young children.

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## TABLE OF CONTENTS

# CHAPTER 1:
# Introduction

## 1.1 Motivation for the Research

Children's dietary habits and food preferences are established early in life (Birch et al., 2007). In the first 2 years after birth, children learn their likes and dislikes, the contexts and consequences of eating, and social influences that facilitate their tasting and intake of food (Birch & Doub, 2014). Eating behaviors learned in early childhood can continue into adolescence and adulthood (Ashcroft et al., 2008; Kotler et al., 2001). Additional work is needed to identify the early-life contributors related to nutrition that promote healthy dietary patterns, growth, and development in young children (Schwarzenberg et al., 2018).

The *Dietary Guidelines for Americans* (DGA) provide nutrition and food-based recommendations to promote health and prevent disease among individuals ages 2 years and old (U.S. Department of Health and Human Services [DHHS] and U.S. Department of Agriculture [USDA], 2015). Currently, the DGA does not provide comprehensive dietary guidance for women during pregnancy or infants and toddlers under age 2 years. To support the inclusion of women who are pregnant and infants and children from birth to 24 months of age in the DGA, the USDA and DHHS initiated the "Pregnancy and Birth to 24 months project", also known as "P/B-24" (Altman et al., 2015; Obbagy et al., 2014). One of the priority topics developed by P/B-24 working groups is the "microenvironmental" effects on the transition to an adult diet during the period of transitional feeding (12-24 months of age), including the identification of factors from the child or the parent that precipitate or exacerbate picky eating behavior (Raiten et al., 2014). Research is needed to develop a better understanding of the etiology of children's picky eating behavior during their transition from a milk-based diet to sharing foods with the family during the first 2 years of life.

Picky eating behavior in children is characterized by the rejection of familiar or novel foods and the consumption of a limited dietary variety, which often interferes with the parent-child relationship during mealtimes (Harris et al., 2018; Taylor et al., 2015; Trofholz et al., 2017). These problematic mealtime behaviors are common in young children, particularly toddlers (Carruth et al., 2004a), and can continue in school-age years and adulthood (Mascola et al., 2010; Van Tine et al., 2017). Research indicates that picky eating behavior is associated with multiple health issues, including emotional and behavioral problems (Machado et al., 2016), psychopathological symptoms (Zucker et al., 2015), eating disorders (Marchi & Cohen, 1990), weight imbalance (Brown et al., 2016b), unhealthy dietary intake (van der Horst et al., 2016), and nutrient deficiencies (Taylor et al., 2016).

Studies have hypothesized that genetics contribute to picky eating in young children (Faith et al., 2013; Fildes et al., 2016), including individual factors related to temperament (Hafstad et al., 2013a; Moding & Stifter, 2017), appetite (Parkinson et al., 2010), and sensory perception (Cole et al., 2017b). In addition, picky eating may be influenced by the feeding environment (Birch, 1999; Galloway et al., 2005), such as aspects related to parent feeding style (Morrison et al., 2013; Podlesak et al., 2017), parent feeding responsiveness (Finnane et al., 2017; Harris et al., 2016; Jansen et al., 2014) and family mealtime routine (Finnane et al., 2017; Hendy et al., 2010). However, the interaction between genetics and parenting has not been investigated in the feeding context and the joint influence on children's eating behaviors is unknown. In addition, little is known about the benefits of a responsive feeding environment on promoting the development of healthy eating behaviors in children as they transition to table foods during the first 2 years after birth (Birch & Doub, 2014; Black & Aboud, 2011; DiSantis et al., 2011; Hodges et al., 2013).

The vast majority of research on picky eating has focused on older children, with less emphasis on children 2 years of age and under (Birch & Doub, 2014; Cole et al., 2017a). The status quo as it pertains to picky eating research is limited to studies that are cross-sectional or conducted in a laboratory setting (Hurley et al., 2011; Taylor et al., 2015). In addition, most research on parent-child mealtime interactions and child eating outcomes are based on unidirectional self-reported surveys with limited attention to genetic risk (Bergmeier et al., 2015a). This dissertation research represents a substantive departure from the status quo by observing parent feeding practices in the naturalistic home environment and examining the prospective effect and interaction with genetic risk on picky eating behavior in children 24 months of age.

### 1.2 Objective

The ***long-term goal*** of this research is to inform the development of evidence-based recommendations and interventions that promote optimal nutrition and weight gain trajectories in young children. Towards that goal, the ***overall objective*** of this dissertation research was to determine the influence of genetics (nature) and the feeding environment (nurture) on picky eating behavior in children aged 24 months from a longitudinal birth cohort. The ***central hypothesis*** is that risk factors related to genetics and the feeding environment are associated with picky eating behavior in young children. The ***rationale*** of this dissertation research was to gain a better understanding of the early-life contributors of picky eating behavior and factors that promote healthy dietary patterns, growth, and development in early childhood. By utilizing a transdisciplinary gene-environment interaction approach, this dissertation research sheds light on the etiology of picky eating behavior in young children as they transition from a milk-based diet to foods consumed by the rest of the family.

## 1.3 Specific Aims

**Aim 1. Nature—Identify the association between individual genetic variations related to appetite and picky eating behavior in children aged 24 months.**

***Working Hypothesis*.** Picky eating behavior is associated with single nucleotide polymorphisms in genes related to appetite.

**Aim 2. Nurture—Identify the association between reported and observed parental feeding responsiveness with picky eating behavior in children aged 24 months.**

Feeding responsiveness will be assessed in the naturalistic home setting during mealtimes. ***Working Hypothesis*.** Higher parental feeding responsiveness is associated with lower picky eating behavior in children.

**Aim 3. Nature/Nurture—Determine the interaction between cumulative genetic risk and parental feeding responsiveness in the prediction of children's picky eating behavior at 24 months.**

Cumulative genetic risk will be constructed based on summing risk alleles in genes related to appetite. ***Working Hypothesis*.** Lower parental feeding responsiveness is positively associated with picky eating behavior in children, with the effects being more pronounced in children with greater cumulative genetic risk.

Although much research has been conducted on picky eating in children, most studies have focused on preschool or school-aged children. Additionally, previous efforts in picky eating research have lacked a transdisciplinary gene-environment interaction approach in addressing the bidirectional feeding relationship among parent-child dyads. This dissertation fills the gap in picky eating research, specifically among children during the transitional feeding period, and contributes a detailed understanding of how genetic variation and the feeding environment influence picky eating behavior in early childhood. These findings can be used to inform the development of evidence-based recommendations and interventions that promote optimal nutrition and weight gain trajectories in young children.

Within this dissertation, Chapter 2 reviews the literature on picky eating in young children, including a systematic review and meta-analysis on the correlates of picky eating and food neophobia in children 2 years of age and younger. Chapter 3 describes the association between variation in genes related to chemosensory perception and picky eating in preschool-aged children. Chapter 4 explains the prospective association between the home feeding environment (specifically, television use during mealtimes, family mealtime routine, and parent feeding practices) in predicting picky eating in preschool-aged children. Chapter 5 delineates the influence of genetics (nature) on picky eating behavior, specifically genetic variants related to appetite (Aim 1). Chapter 6 includes results on the influence of reported and observed parental feeding responsiveness (nurture) on picky eating behavior (Aim 2). Chapter 7 describes the findings on the joint influence of nature and nurture on picky eating behavior utilizing a gene-environment interaction approach (Aim 3). Finally, Chapter 8 summarizes the major findings of this dissertation and provides directions for future research. Supporting documentation, including genotyping methods and the observational codebook, are included in the Appendices.

# CHAPTER 2:
# Literature Review[1]

## 2.1 Defining Picky Eating

Picky eating, also known as "fussy", "faddy", "choosy", or "selective" eating, is a common mealtime problem in children (Taylor et al., 2015). Although there is no consensus on an operational definition of picky eating in children, the behavior is typically characterized by the rejection of familiar or novel foods to the extent of causing problematic interference with the parent-child relationship during mealtimes (Lumeng, 2005). Other characteristics of picky eating include having strong food likes and dislikes, consuming an inadequate amount or variety of food, taking an excessive amount of time to finish the meal, requiring special meal preparation, and requesting a different food than what was served (Boquin et al., 2014a; Boquin et al., 2014b; Dovey et al., 2008). Another characteristic of picky eating is food neophobia, which is the reluctance to try, or the avoidance of, new foods (Birch & Fisher, 1998; Dovey et al., 2008; Pliner, 1994).

Picky eating occurs more frequently in toddlers compared to infants or children 3-11 years of age (Carruth et al., 2004a; Mascola et al., 2010). Prevalence estimates for picky eating range from 21% to 42% for young children in the United States (Carruth et al., 2004a; Jacobi et al., 2003; van der Horst et al., 2016), with a particularly high incidence and prevalence at 2 years of age (Cole et al., 2017a; Mascola et al., 2010). Picky eating is a relatively stable trait in young children (Carruth & Skinner, 2000; Mascola et al., 2010; Steinsbekk et al., 2017); however, several studies indicate it can persist into adolescence and adulthood (Kotler et al., 2001; Thompson et al., 2015; Van Tine et al., 2017; Wildes et al., 2012).

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[1] A portion of this literature review was previously published in *Nutrition Reviews*, entitled "Correlates of picky eating and food neophobia in young children: a systematic review and meta-analysis".

Picky eating behavior is associated with several health issues, such as emotional and behavioral problems. Research indicates that picky eating is associated with more internalizing behaviors (e.g., being withdrawn and having somatic complaints) and externalizing behaviors (e.g., delinquent and aggressive behavior) (Jacobi et al., 2008; Machado et al., 2016; Micali et al., 2011). In addition, picky eating is associated with psychopathological symptoms, including attention and hyperactivity problems, autism spectrum, disruptive/opposite problems, anxiety, and depression (Jacobi et al., 2008; Machado et al., 2016; Segovia, 2015; Zucker et al., 2015). Picky eating is also associated with sensory sensitivity (Farrow & Coulthard, 2012) and eating disorders (Marchi & Cohen, 1990). Studies indicate that picky eating is associated with risk of both underweight and overweight (Antoniou et al., 2016; Dubois et al., 2007a; Finistrella et al., 2012; Galloway et al., 2005; Tharner et al., 2014). Additionally, picky eating is associated with parent health issues, such as maternal alcohol dependence (Lewinsohn et al., 2005) or drug abuse (Zucker et al., 2015), and a drive for thinness (Jacobi et al., 2008).

Previous research has shown that toddlers consume too few vegetables, meat/alternatives, and cereals, yet too much discretionary food (Byrne et al., 2014; Lioret et al., 2013). According to the 2009-2012 National Health and Nutrition Examination Survey (NHANES), toddlers are at risk for inadequate intake of Vitamins D and E, fiber, and potassium (Ahluwalia et al., 2016). Picky eating can negatively affect children's dietary quality (Carruth et al., 1998), and studies have shown that picky eating is associated with lower intake of fruits (Cooke et al., 2006; Galloway et al., 2005; Jacobi et al., 2008; Taylor et al., 2016), vegetables (Cardona Cano et al., 2015; Cooke et al., 2006; Dubois et al., 2007b; Galloway et al., 2005; Galloway et al., 2003; Jacobi et al., 2008; Taylor et al., 2016; Tharner et al., 2014; van der Horst et al., 2016), whole grains (Cardona Cano et al., 2015; Tharner et al., 2014), rice/pasta/potatoes (Cardona Cano et al.,

2015; Jacobi et al., 2008), meat (Cardona Cano et al., 2015; Cooke et al., 2006; Dubois et al., 2007b; Jacobi et al., 2008; Taylor et al., 2016; Tharner et al., 2014; van der Horst et al., 2016), fish/seafood (Cardona Cano et al., 2015; Jacobi et al., 2008; Taylor et al., 2016; Tharner et al., 2014), and milk/dairy (Jacobi et al., 2008; Taylor et al., 2016). For unhealthy food items, the evidence is mixed. One study found that picky eaters consume more savory snacks and confectionary items (Tharner et al., 2014), whereas other studies have found that picky eaters consume less fat, sweets, and fast food (Cardona Cano et al., 2015; Galloway et al., 2005; Jacobi et al., 2008). Unhealthy dietary patterns may compromise nutrient intake, and picky eating is associated with lower intake of vitamins, minerals, and dietary fiber (Galloway et al., 2005; Taylor et al., 2016; Xue et al., 2015). Although picky eaters consume fewer macro- and micro-nutrients compared to non-picky eaters, their intake is still within the recommended intake levels (Taylor et al., 2016).

Picky eating is most frequently measured using parent or caregiver self-report. One method is to ask parents/caregivers a single question (e.g., "is your child a picky eater?") using a 5-point Likert scale (never to always), then creating a dichotomous variable by combining responses of "sometimes" to "always" to be picky eaters and "never" and "rarely" to be non-picky eaters. Another method is using questionnaires, such as the food fussiness subscale of the Children's Eating Behavior Questionnaire (Wardle et al., 2001), the pickiness subscale of the Child Feeding Questionnaire (Birch et al., 2001), and Child Food Neophobia Scale (Pliner, 1994). Finally, some studies measure picky eating through observational methods (de Barse et al., 2017; Luchini et al., 2017a; Luchini et al., 2017b; Tovar et al., 2016). In this method, parents and children are observed during a mealtime in the laboratory, childcare/school, or home setting, and children's eating behaviors (e.g., food refusals) are coded to characterize picky eating.

## 2.2 Nature: Genetic Contributions to Picky Eating

Picky eating is hypothesized to have genetic contributions, with heritability estimates ranging from 67-69% in adults (Knaapila et al., 2007) and 72-78% in children (Cooke et al., 2007; Faith et al., 2013; Fildes et al., 2016). Research suggests that picky eating is related to individual differences in temperament (Hafstad et al., 2013a; Pliner & Loewen, 1997), appetitive traits (Parkinson et al., 2010; Tharner et al., 2014), and taste perception (Cole et al., 2017b; Kauer et al., 2015). **Table 2.1** summarizes potential genetic variants related to picky eating.

### Genes Related to Child Temperament

Temperament refers to biologically-based differences in innate behavioral style, such as emotional, motor, and attentional reactivity (Rothbart, 2007). Heritability estimates for temperament range from 20-60% (Saudino, 2005). Genetic variation in the dopaminergic system may moderate parental influences on developmental outcomes and the reward pathway by regulating the effectiveness of attention, motivation, learning, and reinforcement (Knafo et al., 2011; Queiroz, 2004). Research has identified associations between child temperament and candidate genes related to dopamine receptors, transporters, and degradation of catecholamines (Papageorgiou & Ronald, 2013).

*Dopamine Receptor D2* (*DRD2*) and *Dopamine Receptor D4* (*DRD4*): The *DRD2* gene encodes the dopamine D2 receptor, which plays a key role in subcortical areas of the brain such as the striatum (Jönsson et al., 1999). A frequently studied variant in *DRD2* is the Taq1 allele (rs1800497), which is associated with children's anxiety and differential responses to parenting (Hayden et al., 2010) as well as eating behavior (Obregon et al., 2017). Another commonly studied *DRD2* variant is the intronic SNP rs2283265, which is implicated in binge eating

disorder (Davis et al., 2012) and emotion dysregulation (Gadow et al., 2014). The *DRD4* gene encodes the dopamine D4 receptor, which is mostly expressed in reward system portions of the brain, such as the prefrontal cortex and amygdala (Xiang et al., 2008). A common *DRD4* variant is the 48-base pair variable number tandem repeat (VNTR) polymorphism in exon III (Van Tol et al., 1992). Compared to shorter variants, the 7-repeat variant is associated with novelty seeking (Ebstein et al., 1996), anger-related negative emotionality (Auerbach et al., 2001), and susceptibility to early environmental risk factors, such as poorer parenting quality and maternal insensitivity (Propper et al., 2007; Sheese et al., 2012; Sheese et al., 2007). Another commonly studied *DRD4* variant is the -521 C/T SNP (rs1800955) located in the promoter region, which has been linked to novelty seeking and "risk taking" behavior (Okuyama et al., 2000; Thomson et al., 2013). Non-maltreated children with the TT genotype were more likely to exhibit higher resilient functioning scores compared to maltreated children with the same genotype, supporting the *DRD4* -521 SNP as a candidate variant for differential susceptibility to environmental risk factors (Cicchetti & Rogosch, 2012).

*Dopamine Transporter 1* (*DAT1*): The *DAT1* gene (also known as *SLC6A3*) encodes for DAT1 proteins in the central nervous system (primarily striatum) and is involved in the reuptake of dopamine from neural synapses (Doucette-Stamm et al., 1995). A frequently studied variant in *DAT1* is a 40-base pair VNTR in the 3'-untranslated region (UTR) of the gene, with the 9-repeat and 10-repeat being the most common alleles (Doucette-Stamm et al., 1995). Variations in *DAT1* (including the susceptibility allele, rs40184) are associated with behavior problems in children (Davies et al., 2015).

*Catechol-O-Methyltransferase* (*COMT*): The *COMT* gene encodes for the COMT enzyme that metabolizes catecholamines, such as dopamine and noradrenaline (Tunbridge et al.,

2006). The most frequently studied variant is the Val$^{158}$Met polymorphism (rs4680), which is associated with COMT activity in the prefrontal cortex (Barnett et al., 2008). Variations in *COMT* are associated with children's stress responsivity and susceptibility to parenting (Armbruster et al., 2012; Sulik et al., 2015). In addition, individual variations in COMT genotypes in children has been linked with caregiver emotion regulation and restrictive feeding practices (Bost et al., 2017).

**Genes Related to Appetitive Traits**

Picky eating is associated with appetitive traits such as high satiety responsiveness, slowness in eating, and low food responsiveness and enjoyment of food (Cardona Cano et al., 2015; Tharner et al., 2014). Satiety responsiveness and slowness in eating is the degree to which a child stops eating or chooses not to initiate eating based on his/her perceived fullness, whereas food responsiveness and enjoyment of food both address the child's general appetite and desire to eat when presented with palatable foods (Carnell & Wardle, 2007). Heritability estimates in children range from 63-84% for satiety responsiveness/slowness to eat and 53-75% for food responsiveness/enjoyment of food (Carnell et al., 2008; Llewellyn et al., 2010). Research has identified associations between appetitive traits and several candidate genes.

*Brain-derived Neurotrophic Factor (BDNF)*: The *BDNF* gene encodes BDNF protein, which plays a role in the control of feeding behavior (Cordeira & Rios, 2011). One study among found an epigenetic association, but not a genetic association, between *BDNF* and satiety responsiveness among African-American girls (Gardner et al., 2015), and another study found a negative association between peripheral BDNF and behavioral problems in preschoolers (Yeom

et al., 2016). One particular variant of interest is rs6265 (Val66Met, G196A), which has been linked to obesity in children (Tuyet et al., 2017).

*Fat Mass and Obesity-Associated Protein (FTO)*: The *FTO* gene is predominantly expressed in areas of the hypothalamus related to feeding (Stratigopoulos et al., 2008), and variation in *FTO* is associated with satiety responsiveness, enjoyment of food, and food/energy intake in children (Cecil et al., 2008; Wardle et al., 2008; Wardle et al., 2009). The SNP rs8057044 has been associated with measures of adiposity in African-American adolescents (Bollepalli et al., 2010) and Mexican young adults (Teran-Garcia et al., 2013).

*Ghrelin (GHRL)*: The *GHRL* gene encodes the ghrelin-obestatin neuroprotein that is cleaved into ghrelin and obestatin (Zhang et al., 2005). Ghrelin is known as the "hunger hormone" that is secreted by the stomach and plays a role in energy homeostasis and appetite regulation (Klok et al., 2007). Obese children have been found to have attenuated changes in ghrelin in response to a meal when compared to normal weight children (Nguo et al., 2016). Several *GHRL* SNPs have been associated with obesity-related phenotypes in children, including rs696217 (Leu72Met) and the promoter polymorphism rs27647 (del Giudice et al., 2004; Gueorguiev et al., 2009; Hinney et al., 2002).

*Leptin (LEP) and Leptin Receptor (LEPR)*: The *LEP* gene encodes the hormone leptin, which binds to the leptin receptor (encoded by *LEPR* gene), a single-transmembrane domain receptor of the cytokine receptor family (Tartaglia, 1997). Leptin is known as the "satiety hormone" that is secreted by adipose cells and regulates energy balance by inhibiting hunger (i.e., leptin opposes the actions of ghrelin) (Klok et al., 2007). Obese children, particularly girls, have been found to have high concentrations of serum leptin (Hassink et al., 1996). Additionally, serum leptin concentrations are associated with parent-reported nutritional risk in preschool-aged

children (Persaud et al., 2017). High leptin levels are also associated with emotional eating in boys and stress in girls (Michels et al., 2017).

*Melanocortin 4 Receptor (MC4R)*: The *MC4R* gene on chromosome 18 encodes MC4R, a G protein-coupled receptor primarily expressed in the brain (Mountjoy et al., 1994). MC4R deficiency is a common form of monogenic obesity, and research has indicated that MC4R is involved in feeding behavior and appetite regulation (Adan et al., 2006). Among children, genetic variation in *MC4R* is associated with several appetitive traits, including satiety responsiveness, enjoyment of food, energy intake, and food responsiveness (Cole et al., 2010; Obregón et al., 2017; Valladares et al., 2010). One of the most widely studied loci in obesity research is the SNP rs17782313, which has shown significant associations with height-for-age Z scores (Wang et al., 2013).

*Peptide YY (PYY)*: The *PYY* gene encodes the gastrointestinal hormone Peptide YY (also known as peptide tyrosine tyrosine). Peptide YY is a 36-amino acid member of the neuropeptide Y family of peptides that is released by gastrointestinal cells in response to feeding (le Roux & Bloom, 2005). After a meal, Peptide YY is released into circulation and binds to hypothalamic receptors in the brain to reduce appetite and food intake, and slow gastric emptying (Batterham & Bloom, 2003). Among children, lower levels of PYY in response to a meal have been associated with obesity (Nguo et al., 2016; Roth et al., 2005).

*Genes Related to Reward Pathway*: Genetic polymorphisms relating to reward function may also influence appetitive traits (Stice et al., 2010). Dopamine modulates motivation and reward circuits, and individuals with obesity may have dopamine deficiencies that influence reward sensitivity (Volkow et al., 2011; Wang et al., 2001). Such genes of interest (*DRD2*, *DRD4*, and *COMT*) have been discussed in the previous section.

**Genes Related to Taste Perception**

Children are born with a natural preference towards sweet and salty tastes, and aversions to sour and bitter tastes (Birch, 1999; Drewnowski, 1997). Genetic variation in taste perception may contribute to differences in food preferences and eating behavior (Cooke et al., 2007; Faith et al., 2013; Feeney et al., 2011; Fildes et al., 2016; Fildes et al., 2014), and heritability estimates for food preferences in children range from 20-78% (Breen et al., 2006). Research on taste perception and genetic variation has focused primarily on sweet or bitter taste in the Carbonic Anhydrase 6 gene (*CA6*), G protein subunit alpha transducin 3 gene (*GNAT3*), Taste Receptor Type 1 Member 2 (*TAS1R2*) and Member 3 genes (*TAS1R3*), Taste Receptor Type 2 Member 1 (*TAS2R1*) and Member 19 genes (*TAS2R19*), and Taste Receptor Type 2 Member 38 gene (*TAS2R38*) (Bachmanov & Beauchamp, 2007; Hayes et al., 2013; Mennella et al., 2016).

*Genes Related to Sweet Taste: TAS1R2, TAS1R3, GNAT3*: The sweet taste receptor in humans is encoded by *TAS1R2* and *TAS1R3* genes located on chromosome 1 (Liao & Schultz, 2003). The sweet taste receptor requires the heterodimeric combination of TAS1R2 and TAS1R3, which are two G protein-coupled receptors in the taste receptor type 1 family (Keskitalo et al., 2007). The *GNAT3* gene encodes gustducin, a G protein-associated with intracellular sweet signaling (Reed & Margolskee, 2010). One variant of interest is rs7792845, which has been linked with better ability to sort low concentrations of sucrose (Fushan et al., 2010). Genetic variation for sweet taste receptors and gustducin have been associated with differences in sweet taste perception and preferences (Dias et al., 2015; Fushan et al., 2009; Ramos-Lopez et al., 2016). In a study involving participants from several different countries, the *TAS1R2* gene was positively associated with liking of white wine and vodka, and a diminished liking for lamb meat (Pirastu et al., 2012).

*Genes Related to Bitter Taste: TAS2R1, TAS2R19, TAS2R38 and CA6*: Receptors in the taste receptor type 2 family function as bitter taste receptors, and these receptors are encoded by genes primarily on chromosomes 7 and 12 (Chandrashekar et al., 2000). For *TAS2R1*, one particular SNP of interest is rs2234233, which has been implicated in glucose homeostasis (Dotson et al., 2008). For *TAS2R19*, the variant rs10772420 encodes for either cysteine or arginine on the bitter taste receptor, and the cysteine version is associated with more sensitivity to the bitterness of quinine and grapefruit juice (Hayes et al., 2015).

The most studied bitter receptor gene is *TAS2R38* on chromosome 7, which is associated with human phenylthiocarbamide (PTC) bitter taste sensitivity and 6-n-propyl-2-thiouracil (PROP) bitterness perception (Keller et al., 2002; Mennella, 2005; Turnbull & Matisoo-Smith, 2002). Sensitivity to PROP and PTC is related to three functional variants in the *TAS2R38* that encode for amino acid substitutions: rs713598, rs1726866, and rs10246939 (Kim, 2003). These three SNPs contribute to two common haplotypes related to detection thresholds for bitter taste: the dominant taster haplotype (PAV, Pro-Ala-Val) confers bitter sensitivity and the recessive non-taster haplotype (AVI, Ala-Val-Ile) is less functional (Bufe et al., 2005; Kim, 2003). The SNPs rs1726866 and rs10246939 are related to sucrose detection thresholds in children (Joseph et al., 2016), and rs713598 has shown associations with picky eating (Cole et al., 2017b).

Another gene related to bitter sensitivity is *CA6* (also known as gustin), which may influence taste bud development and bitter taste perception (Calò et al., 2011; Melis et al., 2013; Padiglia et al., 2010). The gustin polymorphism, rs22274333, is involved in taste bud growth and development (Barbarossa et al., 2015; Melis et al., 2013). Additionally, previous studies have shown associations between *CA6* rs227432 and picky eating (Cole et al., 2017b).

## 2.3 Nurture: Feeding Environment and Picky Eating

Picky eating behavior may lead parents to adapt inappropriate feeding practices to change their child's food intake. Since picky eaters often have limited dietary intake, parents may worry about undereating (Brown et al., 2016a) and utilize specific feeding practices in order to influence their child's food preferences (Russell et al., 2015). In terms of the feeding environment, there is a division of responsibility in that parents are responsible for what, when, and where children eat, while children are responsible for how much and whether to eat (Danaher & Fredericks, 2012; Satter, 1990). Parents are the gatekeepers of children's food intake during infancy and toddlerhood by influencing *what* and *when* children eat (e.g., home food availability and the presence and frequency of family mealtimes) and *how* they are fed (i.e., feeding responsiveness and feeding practices) (Birch & Davison, 2001).

### Parent Feeding Responsiveness

Feeding responsiveness is the extent that parents foster self-regulation by exhibiting warmth and attunement (Eneli et al., 2008), and their ability to recognize, interpret, and respond appropriately to the child's signals of hunger and satiety (Hodges et al., 2013). Responsive feeding is a strategy parents can use to help children learn to like healthy food during the 12 to 24 month transitional feeding period (Pérez-Escamilla et al., 2017). Responsive feeding is prompt, emotionally supportive, and developmentally appropriate, and facilitates the child's hunger and satiety cues, establishes routines and structure around mealtimes, and promotes self-regulation (Black & Aboud, 2011; DiSantis et al., 2011). Examples of responsive feeding include child-centered feeding practices that promote child autonomy, such as reasoning, modeling, and monitoring. Nonresponsive feeding lacks reciprocity between the parent and child, does not

establish eating in the same place, does not provide food that is healthy and developmentally appropriate, and overrides the child's hunger and satiety cues (Black & Aboud, 2011; DiSantis et al., 2011). Examples of nonresponsive feeding includes parent-centered feeding practices that attempt to control children's eating behaviors, such as control in feeding, pressure to eat, and restriction. If feeding is nonresponsive, then children may resort to picky eating behaviors that attract caregiver/parent attention, such as refusing to eat (Black & Aboud, 2011). There is strong evidence indicating that nonresponsive feeding practices are associated with picky eating in children (Birch & Fisher, 1998; Carruth et al., 1998; Finnane et al., 2017; Galloway et al., 2005; Galloway et al., 2003; Morrison et al., 2013; Tharner et al., 2014; Webber et al., 2010).

**Early Feeding Practices**

Feeding practices are behaviors or actions (intentional or unintentional) that parents use to influence children's food intake (Vaughn et al., 2013) (Vaughn et al., 2016). During infancy, parents influence the feeding environment through early feeding practices, such as the mode and duration of milk feeding (i.e., breastfeeding, formula-feeding, or feeding a combination of breastmilk and formula) and the timing and type of complementary food during the introduction to solids. The American Academy of Pediatrics recommends exclusive breastfeeding for at least 6 months, and the introduction of solid foods at 4 to 6 months (Kleinman, 2000). Previous studies suggest that exclusive breastfeeding for 6 months and the introduction of complementary and solid foods after 6 months is associated with a lower odds of picky eating (Galloway et al., 2003; Northstone et al., 2001; Shim et al., 2011). However, the evidence between early feeding practices and picky eating remains unclear and additional research is needed (Cassells et al., 2014; de Barse et al., 2017; Ünlü et al., 2008).

## Family Mealtime Routine

Family mealtime routine refer to the parent-created structure, location, timing, and atmosphere of meals (Fiese et al., 2006; Vaughn et al., 2013). One of the most frequently studied family mealtime routine is eating dinner together as a family (Bekelman et al., 2017). A structured meal environment ensures a pleasant feeding context with routines and supportive limits around mealtimes by reducing distractions, having a predictable schedule (i.e., eating in the same place at the same time), ensuring child is seated in a supportive and comfortable position, and providing food that is tasty, healthy, and developmentally appropriate (Eneli et al., 2008; Satter, 1995). Structured family meals are negatively associated with picky eating (Jansen et al., 2014). Other benefits of family meals include fewer behavior problems (Hofferth & Sandberg, 2001), improved vitamin and mineral intake (Neumark-Sztainer et al., 2003), and better weight outcomes (Hammons & Fiese, 2011).

## Home Food Availability

Home food availability refers to the presence or absence of healthy and unhealthy foods in the home and whether the food is permissible for the child to eat (Fulkerson et al., 2008; Holsten et al., 2012; Vaughn et al., 2013). Children's dietary patterns are influenced by the foods that parents make available in the home (Birch & Davison, 2001). According to one qualitative study on the process of children's food choices at home, food availability was a primary environmental influence on children's food options and consumption (Holsten et al., 2012). Children's fruit and vegetable consumption is positively associated with the availability of fruits and vegetables in the home (Fulkerson et al., 2010; Shim et al., 2016), and children who have sugar-sweetened beverages at home have lower diet quality (Santiago-Torres et al., 2014).

## 2.4 Nature-Nurture: Gene-Environment Interaction and Picky Eating

The bioecological model, first proposed by Bronfenbrenner and Ceci (1994), highlights the importance of understanding gene-environment interactions in human development. Gene-environment interaction (GxE) is the joint effect of one or more genes with one or more environmental factors that cannot be readily explained by their independent effects (Dick, 2011; Thomas, 2010). The GxE approach has been used extensively in research related to the development of human diseases (Hunter, 2005) and behavior, particularly psychological traits and disorders (Manuck & McCaffery, 2014). Among children, GxE approaches have be used to understand the joint influence of genetics (nature) and parent influences (nurture) on child outcomes (Collins et al., 2000; Meaney, 2010; Sameroff, 2010). Genetic risk may interact with the rearing environment to predict children's externalizing behavior, self-regulation, social and emotional development, and temperament (Bakermans-Kranenburg & Van Ijzendoorn, 2006; Berry et al., 2014; Luijk et al., 2011; Sheese et al., 2007). In addition, research indicates that the interplay between nature and nurture contributes to children's food preferences, dietary habits, and energy intake (Butte et al., 2006; Cooke et al., 2007; Fildes et al., 2014; Hetherington & Cecil, 2010; Wardle & Cooke, 2008). However, no study has been conducted on picky eating in children using a GxE approach.

GxE on child behavior can be conceptualized by two different models: the diathesis stress (or dual-risk) model and the differential susceptibility hypothesis. The diathesis stress model suggests that due to genetic variation some individuals may be more vulnerable to adversity (**Fig. 2.1A**), whereas the differential susceptibility hypothesis holds that genetic variants may confer plasticity to environmental influences in a "for better and for worse" manner and enhance sensitivity to both supportive and negative conditions (Belsky et al., 2007; Belsky & Pluess,

2009; Boyce & Ellis, 2005). Thus, based on the differential susceptibility hypothesis, children who are genetically susceptible to environmental influences display are pickier eaters compared to children who are not genetically susceptible. Similar to the differential susceptibility hypothesis, the biological sensitivity to context hypothesis posits that individuals vary in their susceptibility to environmental influences, and additionally describes children as orchids when they are highly reactive to the environment or dandelions if they a less reactive and more resistant to environmental influences (Ellis & Boyce, 2008). Thus, according to the differential susceptibility framework with the orchid-dandelion hypothesis (**Fig. 2.1B**), children who are genetically susceptible to environmental influences (i.e., orchid children) will display more negative outcomes in unfavorable environments and more positive outcomes in favorable environments, when compared to children who are less genetically susceptible to environmental influences (i.e., dandelion children) (Belsky et al., 2009; Boyce & Ellis, 2005; Ellis & Boyce, 2008). Based on this framework, orchid children are non-picky eaters when exposed to a responsive feeding environment, but are picky eaters in a nonresponsive feeding environment. These results would be more pronounced compared to dandelion children.

GxE on behavior has typically been studied one genetic polymorphism at a time (Burmeister et al., 2008). However, combining the effects of multiple genes provides stronger evidence of genes moderating environmental effects (Sonuga-Barke et al., 2009). Genetic risk scores provide a summary of risk-associated variation by aggregating information from multiple genetic variants (Horne et al., 2005). Measuring the cumulative effects of genetic variants has been conducted in research studies on genes related to appetite (Li et al., 2010), temperament (Belsky & Beaver, 2011), but not taste perception.

## Correlates of Picky Eating and Food Neophobia in Young Children: A Systematic Review and Meta-Analysis[2]


### Abstract

**Context:** Picky eating behavior is prevalent among toddlers and may negatively impact their growth and development.

**Objective:** This article summarizes the correlates of picky eating and food neophobia in young children, which were identified using a socio-ecological framework.

**Data Sources:** A literature search was conducted in 4 electronic databases.

**Study Selection:** Inclusion criteria were English-language peer-reviewed publications that investigated correlate(s) of picky eating or food neophobia in children aged ≤ 30-months.

**Data Extraction:** Correlates were categorized into 4 levels: cell, child, clan (family), and community/country. Thirty-two studies, which examined 89 correlates, were identified from the keyword searches of the databases and manual searches of the reference lists of included articles.

**Results:** The most examined correlates were characteristics related to the child (sex, weight, and dietary intake) and parent (feeding beliefs and practices). A meta-analysis estimated the prevalence of picky eating to be 22%. Each additional month of a child's age was associated with a 0.06 U increase in the Children's Eating Behavior Questionnaire food fussiness score.

**Conclusion:** This review highlights the importance of investigating child-parent dyads and bidirectional feeding interactions and draws attention to the lack of picky eating research at the level of the cell and the community/country.


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[2] This article appeared in its entirety as Cole, N. C., An, R., Lee, S. Y., & Donovan, S. M. (2017). Correlates of picky eating and food neophobia in young children: a systematic review and meta-analysis. *Nutrition Reviews*, *75*(7), 516-532. This article is reproduced by permission of Oxford University Press (https://global.oup.com)

## 2.5 Introduction

Picky eating is characterized by low dietary variety, unwillingness to eat either familiar or novel foods, and problematic interference with the parent-child relationship (Boquin et al., 2014a; Lumeng, 2005). These behaviors are prevalent in young children – up to one half of toddlers are reported to be picky eaters (Carruth et al., 2004a). One longitudinal study showed that the highest incidence of picky eating occurred in children two years of age (Mascola et al., 2010). The first two years of life are critical in the development of healthy eating habits (Birch & Doub, 2014; Cashdan, 1994), and dietary patterns and food preferences established in early life continue in preschool, school-aged years and young adulthood (Nicklaus et al., 2005; Siega-Riz et al., 2010; Skinner et al., 2002). During infancy and toddlerhood, children develop autonomy, an emerging sense of self (such as self-concept), social competence, and self-regulation (Houck, 1999). Aligning these developmental changes is the ability to control positive and negative emotions (Kopp, 1989) and comply with caregivers' requests (Kochanska et al., 2001). Feeding difficulties often emerge in young children as they acquire skills related to gross motor development and self-feeding, and they transition from a liquid-based diet to foods consumed by the family (Birch & Doub, 2014; Carruth et al., 2004b). Picky eating behavior may persist later in life (Ashcroft et al., 2008; Kotler et al., 2001; Marchi & Cohen, 1990; Thompson et al., 2015; Wildes et al., 2012), and additional research is needed to develop a better understanding of the early life factors contributing to picky eating behavior during the transition to an adult diet.

Picky eating can affect children's nutrition intake and dietary quality, and as a consequence, negatively impact their growth and development (Cardona Cano et al., 2016; Dubois et al., 2007a). Studies have also shown associations between picky eating and risk of depression (Zucker et al., 2015), eating disorders (Marchi & Cohen, 1990), and emotional and

behavioral problems (Machado et al., 2016). Although picky eating behavior has been hypothesized to be related to genetic contributions (Fildes et al., 2016) and environmental factors (Dubois et al., 2007a; van der Horst, 2012), the developmental pathway of picky eating in early childhood is largely unknown (Harris, 2000). Moreover, the vast majority of research assessing picky eating behavior focuses on preschoolers (children 2-5 years of age), whereas much less attention has been paid to children 2 years of age and younger. Unlike preschoolers, infants and toddlers are completely dependent on parents and caregivers to feed them, and are learning how to eat through familiarization, observation, and associative learning (Birch & Doub, 2014). Previous reviews have addressed picky eating in terms of prevalence estimates, assessment methods, and relation to dietary intake, parent-child interactions, cognition, and social factors (Cano et al., 2015; Lafraire et al., 2016; Taylor et al., 2015). While these studies provide an extensive review of picky eating, the evidence on children 2 years of age and younger is still unclear. To our knowledge, a systematic review and meta-analysis of the multi-level correlates of picky eating in this young child population has not been conducted.

The objective of this study was to systematically review existing scientific evidence of correlates of picky eating and food neophobia in children aged ≤2 years. The authors adopted a socioecological model as a theoretical framework to examine picky eating research (Harrison et al., 2011). Correlates were classified into 4 broad categories—namely, cell (genetic and biological factors), child (individual characteristics of the child), clan (familial and parental factors), and community/country (factors outside of the home). Use of a socioecological framework to classify study findings into major categories allows for a better understanding of the context of picky eating behavior and highlight areas where additional research is warranted. Through the investigation of multidimensional factors of picky eating, this review also aids the

development of evidence-based recommendations and interventions to promote healthy eating patterns and weight-gain trajectories in young children.

## 2.6 Methods

### Study Selection Criteria

Systematic review and meta-analysis procedures were conducted in accordance with the *Cochrane Handbook for Systematic Reviews of Interventions* and Preferred Reporting Items for Systematic Reviews and Meta-Analyses (see **Table 2.5** in the Supporting Information online) (Higgins & Green, 2008; Liberati et al., 2009). The PICO (population, intervention, comparison, outcomes) framework (**Table 2.2**) was established *a priori* to formulate the systematic research question and determine the inclusion and exclusion for studies. Studies that met both of the following criteria were included in the review: (1) presented a quantitative analysis of at least 1 correlate of picky eating behavior or food neophobia in children aged ≤30 months and, (2) was published in English in a peer-reviewed publication. The age ceiling of 30 months rather than 24 months was imposed to retain studies that included a sample with children slightly older than 2 years. Some authors argue that children aged ≤12 months may be too young to exhibit a true reflection of picky eating (Taylor et al., 2015). Because picky eating assessment is often based on parental perception (Taylor et al., 2015) and complementary feeding often begins in the first year of life (Birch & Doub, 2014), studies on children aged ≤12 months were included in this review. There were no restrictions on publication dates or type of study design. Longitudinal studies were included if the data presented in the article were consistent with the age limits (e.g., if the study examined participants at aged 2 years and again at age 7 years, only baseline measurements at age 2 years were used). Articles were excluded from the review if they met any

of the following criteria: not published in English; not peer reviewed (e.g., dissertation or conference proceeding); review paper or case study; reported a study done exclusively on animals or adult subjects; or reported a study that evaluated only children with chronic diseases, developmental disability, autism spectrum disorder, or avoidant/restrictive food intake disorder. Although review papers were not eligible for inclusion, relevant review articles were obtained and their reference lists were searched for eligible articles and to compare findings. If multiple publications were available from the same data source or study population, only the study with the most complete or recent data was included to prevent duplication. If the studies contained different information, then both were included.

**Search Strategy**

Keyword search was performed in 4 electronic bibliographic databases: PubMed, PyscINFO, Cochrane Library, and Web of Science. The search algorithm included all possible combinations of keywords from the following two groups: (1) “child”, “toddler”, “infant”, “baby”, or “babies”; and (2) “picky eating”, “pickiness”, “fussy eating”, “choosy eating”, “faddy eating”, “food neophobia”, “food fussiness”, or “selective eating”. Titles and abstracts of the articles identified through the keyword search were screened against the study selection criteria by a single author (N.C.). Disagreement about study eligibility was solved through discussion with the co-authors (R.A., S.L., and S.D.). Potentially relevant articles were retrieved for evaluation of the full text by a single author (N.C.).

A reference list search (i.e., backward reference search) and cited reference search (i.e., forward reference search) were conducted based on the full-text articles meeting the study selection criteria that were identified from the keyword search. Articles identified from the backward and forward reference search were further screened and evaluated using the same

study selection criteria. Reference searches were repeated on all newly identified articles until no additional relevant article was found. The last search was completed on July 13, 2016.

**Data Extraction**

A standardized data extraction form was used to collect the following methodological and outcome variables from each included study: author(s), publication year, country of study, study design, sample size, age of subjects, measurement tool(s) for assessing picky eating, method of reporting picky eating (i.e., maternal report, parent report, etc.), prevalence of picky eating or food neophobia, specific picky eating behavior(s) of interest, and result(s) on the relationship of correlate(s) to picky eating. Data from included studies was extracted by one author (N.C.) and verified by a second author (R.A.). If there was missing data, then the corresponding author was contacted. Analysis included a narrative review of the correlates classified into four levels: cell, child, clan/family, and community/country. Food neophobia is often considered a subset of picky eating, and corresponding behaviors are frequently measured as two separate constructs because there is no clear distinction between them.(Lafraire et al., 2016; Pelchat & Pliner, 1986) Given that studies examining both picky eating and food neophobia were included in this review, the term *picky eating* is used when discussing overall findings, but *food neophobia* is specified when it was specifically measured.

**Quantitative Data Synthesis**

Meta-analysis was conducted to estimate (1) the prevalence of picky eating based on five studies that adopted a dichotomous definition for picky eating (i.e., picky or not), and (2) the degree of picky eating based on six studies that adopted a continuous measure of picky eating, namely the food fussiness subscale in the Children's Eating Behavior Questionnaire (CEBQ). Due to data availability and measurement heterogeneity, additional meta-analyses could not be

conducted on other covariates of picky eating such as sex, weight, and diet. Study heterogeneity was assessed using the $I^2$ index. A random-effect model was estimated given an $I^2$ statistic at or above 50%; otherwise, a fixed-effect model was estimated. Publication bias was assessed by visual inspection of the funnel plot, and Begg's and Egger's tests. Meta-regression was performed to assess the relationship between the mean age of study sample and the CEBQ food fussiness score. All statistical analyses were conducted using Stata 14.1 SE version (StataCorp, College Station, TX). Statistical significance was set to p-value $< 0.05$.

**Study Quality Assessment**

The quality of included studies was assessed by the following six criteria based on the quality assessment scales from the *Methods Guide for Effectiveness and Comparative Effectiveness Reviews* developed by the Agency for Healthcare Research and Quality (AHRQ), the National Heart, Lung, and Blood Institute (NHLBI), and methods previously used (Pearson & Biddle, 2011): (1) *a priori* aim/hypothesis specific to picky eating behaviors; (2) study population clearly specified and defined; (3) study design; (4) sample size; (5) reliable and valid measure of picky eating behavior; and (6) well-validated measure of correlate(s). Scores for each criterion range from 0 to 2, depending on whether the criterion was unmentioned or unmet (0), partially met (1), or completely met (2). For *a priori* aim/hypothesis, studies that explicitly stated picky eating behaviors were given a score of 2, studies that implied picky eating were given a score of 1 (e.g., eating difficulties), and studies that did not indicate aims/hypothesis specific to picky eating were given a score of 0. For study design, randomized controlled trials (RCTs) were given a score of 2, cohort studies a score of 1, and cross-sectional studies a score of 0. For sample size, study samples $\geq 1000$ were given a score of 2, samples 100-999 a score of 1, and samples $< 100$ a score of 0. A total quality score ranging from 0-12 was obtained for each study

by summing the score of each criterion. The overall study quality score helped measure the strength of the study evidence but was not used to determine the inclusion of studies in the review.

## 2.7 Results

### Study Selection

**Fig. 2.2** shows the flowchart of study selection and reasons for exclusion. A total of 747 unduplicated articles were identified through keyword and reference search, among which 622 were excluded in the title/abstract screening. The remaining 125 articles were reviewed in full text against the study selection criteria. Of these, 32 articles met inclusion criteria and were included in the review.

### Basic Characteristics of the Included Studies

**Table 2.2** summarizes the basic characteristics of the 32 articles included in the review. More than half of the studies (n=22) were published from 2010 onward (Barends et al., 2014; Brown & Lee, 2015; Cao et al., 2012; Cardona Cano et al., 2015; Cassells et al., 2014; Fildes et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Jansen et al., 2014; Johnson et al., 2016; Magarey et al., 2016; Maslin et al., 2015; McCarthy et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Northstone & Emmett, 2013; Oliveira et al., 2015; Perry et al., 2015; Quah et al., 2015; Svensson et al., 2011; Syrad et al., 2015; Yuan et al., 2016). Regarding study location, 20 were conducted in Europe [UK (Brown & Lee, 2015; Fildes et al., 2015; Fletcher et al., 2016; Johnson & Harris, 2004; Johnson et al., 2016; Maslin et al., 2015; Northstone & Emmett, 2013; Northstone et al., 2001; Syrad et al., 2015; Wright et al., 2007); France (Migraine et al., 2013; Monnery-Patris et al., 2015; Oliveira et al., 2015; Yuan et al., 2016); Ireland (Blossfeld et al.,

2007a; Blossfeld et al., 2007b; McCarthy et al., 2015); Netherlands (Barends et al., 2014; Cardona Cano et al., 2015); Sweden (Svensson et al., 2011)]. A total of five studies were conducted in Australia (Cassells et al., 2014; Howard et al., 2012; Jansen et al., 2014; Magarey et al., 2016; Perry et al., 2015); four in the US (Briefel et al., 2006; Carruth et al., 2004a; Hendricks et al., 2006; Reau et al., 1996); one in China (Cao et al., 2012); one in Singapore (Quah et al., 2015); and one in Canada (Sauve & Geggie, 1991). In terms of study design, 20 papers were cross-sectional (Blossfeld et al., 2007a; Blossfeld et al., 2007b; Briefel et al., 2006; Cao et al., 2012; Carruth et al., 2004a; Fildes et al., 2015; Fletcher et al., 2016; Hendricks et al., 2006; Howard et al., 2012; Jansen et al., 2014; Johnson & Harris, 2004; Johnson et al., 2016; Maslin et al., 2015; McCarthy et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Northstone & Emmett, 2013; Perry et al., 2015; Reau et al., 1996; Wright et al., 2007); 11 papers were longitudinal studies (Barends et al., 2014; Brown & Lee, 2015; Cardona Cano et al., 2015; Cassells et al., 2014; Northstone et al., 2001; Oliveira et al., 2015; Quah et al., 2015; Sauve & Geggie, 1991; Svensson et al., 2011; Syrad et al., 2015; Yuan et al., 2016); and one paper was an RCT (Magarey et al., 2016). The sample sizes ranged from a small-scale preliminary study (n=18) (Johnson & Harris, 2004) to a large longitudinal cohort (n=9599) (Northstone & Emmett, 2013). Five studies had a sample size of less than 100 participants (Barends et al., 2014; Blossfeld et al., 2007a; Blossfeld et al., 2007b; Johnson & Harris, 2004; Svensson et al., 2011); 15 studies had a sample size of 100-999 (Brown & Lee, 2015; Cao et al., 2012; Cassells et al., 2014; Fletcher et al., 2016; Howard et al., 2012; Jansen et al., 2014; Magarey et al., 2016; Maslin et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Perry et al., 2015; Quah et al., 2015; Reau et al., 1996; Sauve & Geggie, 1991; Wright et al., 2007); and 12 studies had a sample size above 1000 (Briefel et al., 2006; Cardona Cano et al., 2015; Carruth et al., 2004a;

Fildes et al., 2015; Hendricks et al., 2006; Johnson et al., 2016; McCarthy et al., 2015; Northstone & Emmett, 2013; Northstone et al., 2001; Oliveira et al., 2015; Syrad et al., 2015; Yuan et al., 2016). The majority of studies (n=24) included children during the transitional 12-24 month feeding period. However, some studies also included children younger than 12 months (Briefel et al., 2006; Carruth et al., 2004a; Hendricks et al., 2006; Sauve & Geggie, 1991) or older than 24 months (Fletcher et al., 2016; Jansen et al., 2014; Maslin et al., 2015; Reau et al., 1996; Wright et al., 2007). In addition, three studies compared children who had been delivered pre-term to those born full-term (Johnson et al., 2016; Migraine et al., 2013; Sauve & Geggie, 1991).

Of the 32 studies included in the review, 21 were on picky eating (i.e., food fussiness, choosy eating, faddy eating, and fussy eating) (Blossfeld et al., 2007a; Briefel et al., 2006; Brown & Lee, 2015; Cao et al., 2012; Cardona Cano et al., 2015; Carruth et al., 2004a; Fildes et al., 2015; Hendricks et al., 2006; Jansen et al., 2014; Johnson et al., 2016; Magarey et al., 2016; Maslin et al., 2015; McCarthy et al., 2015; Northstone & Emmett, 2013; Northstone et al., 2001; Quah et al., 2015; Reau et al., 1996; Sauve & Geggie, 1991; Svensson et al., 2011; Syrad et al., 2015; Wright et al., 2007); eight were on food neophobia (Cassells et al., 2014; Fletcher et al., 2016; Howard et al., 2012; Johnson & Harris, 2004; Monnery-Patris et al., 2015; Oliveira et al., 2015; Perry et al., 2015; Yuan et al., 2016); and three studies investigated both picky eating and food neophobia (Barends et al., 2014; Blossfeld et al., 2007b; Migraine et al., 2013). A variety of methods were used to assess picky eating and food neophobia, yet all studies relied on caregiver report (primarily the mother). There were 9 studies that used a single question (e.g., “is your child a picky eater?”) (Barends et al., 2014; Briefel et al., 2006; Carruth et al., 2004a; Fletcher et al., 2016; Hendricks et al., 2006; Northstone & Emmett, 2013; Northstone et al., 2001; Reau et

al., 1996; Wright et al., 2007) and two studies that used study-specific questions (Oliveira et al., 2015; Yuan et al., 2016). The majority of studies (n=20) used existing questionnaires, including the Picky Eating Questionnaire (Blossfeld et al., 2007b; Maslin et al., 2015); Eating Behavior Questionnaire (Johnson et al., 2016); CEBQ (Blossfeld et al., 2007a; Blossfeld et al., 2007b; Brown & Lee, 2015; Cao et al., 2012; Fildes et al., 2015; Jansen et al., 2014; Magarey et al., 2016; McCarthy et al., 2015; Quah et al., 2015; Svensson et al., 2011; Syrad et al., 2015); Child Food Neophobia Scale (CFNS) (Cassells et al., 2014; Howard et al., 2012; Perry et al., 2015); Children's Eating Difficulties Questionnaire (Migraine et al., 2013; Monnery-Patris et al., 2015); Child Behavior Checklist (Cardona Cano et al., 2015); and the Reaction to Food scale from the Colorado Childhood Temperament Inventory (CCTI) (Johnson & Harris, 2004). The most commonly used questionnaire was the CEBQ (n=11), which contains a food fussiness subscale that assesses picky eating behavior in children. Of the studies that examined food neophobia, the most commonly used tool was the CFNS (n=3), which assesses a child's willingness to try new foods.

Although the majority of studies used continuous measures of picky eating, several studies shared similar response categories for the frequency of picky eating (i.e., Likert scales that were typically anchored by "never", "sometimes", and "always") and dichotomized picky eating by combining responses of "sometimes" to "always" to be picky eaters and "never" and "rarely" to be non-picky eaters. Out of the 24 studies that investigated picky eating, six reported prevalence estimates of picky eating with sample sizes based on frequency responses and dichotomization (Cardona Cano et al., 2015; Carruth et al., 2004a; Hendricks et al., 2006; Northstone & Emmett, 2013; Reau et al., 1996; Wright et al., 2007). As shown in **Table 2.2**, prevalence estimates ranged from 7%-36% for children aged 4-30 months.

## Correlates of Picky Eating Behaviors

A total of 89 correlates classified into 4 levels (i.e., cell, child, clan, and community/country) were assessed, with the majority (n = 21) of studies assessing 1-4 correlates (Barends et al., 2014; Blossfeld et al., 2007a; Blossfeld et al., 2007b; Briefel et al., 2006; Fildes et al., 2015; Fletcher et al., 2016; Johnson & Harris, 2004; Johnson et al., 2016; Magarey et al., 2016; McCarthy et al., 2015; Monnery-Patris et al., 2015; Northstone & Emmett, 2013; Northstone et al., 2001; Oliveira et al., 2015; Perry et al., 2015; Quah et al., 2015; Reau et al., 1996; Sauve & Geggie, 1991; Svensson et al., 2011; Syrad et al., 2015; Yuan et al., 2016), and the remaining 11 assessing ≥5 correlates (Brown & Lee, 2015; Cao et al., 2012; Cardona Cano et al., 2015; Carruth et al., 2004a; Cassells et al., 2014; Hendricks et al., 2006; Howard et al., 2012; Jansen et al., 2014; Maslin et al., 2015; Migraine et al., 2013; Wright et al., 2007). **Table 2.3** reports a summary of the associations between potential correlates of picky eating and food neophobia.

### *Cell: Genetic and Biological Contributions*

Four studies investigated genetic and biological correlates, such as *in-utero* factors (being born pre-term and gestational age) and medical issues (Hendricks et al., 2006; Johnson & Harris, 2004; Johnson et al., 2016; Migraine et al., 2013). Evidence for picky eating and being born pre-term was mixed in cross-sectional studies. One study found a positive association between being born pre-term and picky eating (Johnson et al., 2016), but another found no association (Migraine et al., 2013). One cross-sectional study found no association between gestational age at birth and picky eating (Migraine et al., 2013). Two cross-sectional studies examined the association between child medical issues and picky eating behaviors (Hendricks et al., 2006; Johnson & Harris, 2004). Illness during late infancy (i.e., vomiting, diarrhea, or constipation)

was positively associated with food neophobia (Johnson & Harris, 2004). There was no association between picky eating and the child's reported allergies (Hendricks et al., 2006), long-term medical problems (Hendricks et al., 2006), or oral-motor-skill function (Johnson & Harris, 2004).

### *Child: Personal Characteristics*

#### *Demographics*

Eleven studies investigated demographic correlates related to the child (Barends et al., 2014; Cao et al., 2012; Cardona Cano et al., 2015; Carruth et al., 2004a; Cassells et al., 2014; Hendricks et al., 2006; Johnson et al., 2016; Maslin et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Svensson et al., 2011). The findings on picky eating in relation to age were mixed for both longitudinal and cross-sectional studies. Age was found to be positively associated with picky eating in 3 studies (Barends et al., 2014; Carruth et al., 2004a; Svensson et al., 2011), but 2 studies found no association (Cassells et al., 2014; Maslin et al., 2015). Nine studies investigated the association between sex and picky eating; 2 were longitudinal studies (Cardona Cano et al., 2015; Cassells et al., 2014), and 2 studies included children aged < 12 months (Carruth et al., 2004a; Maslin et al., 2015). Null findings were reported in general for the association between sex and picky eating; however, 1 cross-sectional study found a positive association among girls (Cao et al., 2012). There was no relationship between picky eating and birth order (Carruth et al., 2004a) or child race/ethnicity (Carruth et al., 2004a).

#### *Anthropometrics*

Twelve studies examined the association between anthropometric measurements and picky eating behaviors (Brown & Lee, 2015; Cao et al., 2012; Carruth et al., 2004a; Cassells et al., 2014; McCarthy et al., 2015; Migraine et al., 2013; Oliveira et al., 2015; Perry et al., 2015;

Quah et al., 2015; Sauve & Geggie, 1991; Wright et al., 2007). There was no association between birth weight and picky eating in cross-sectional (Hendricks et al., 2006; Migraine et al., 2013) or longitudinal studies (Cassells et al., 2014; Oliveira et al., 2015). However, the evidence for current weight was mixed for both longitudinal and cross-sectional studies. Three studies found a negative association between current weight and picky eating (Brown & Lee, 2015; Carruth et al., 2004a; McCarthy et al., 2015), and 6 studies found no association (Cao et al., 2012; Cassells et al., 2014; Perry et al., 2015; Quah et al., 2015; Sauve & Geggie, 1991; Wright et al., 2007). There was no association between picky eating and the child's height (Wright et al., 2007) or weight gain (Quah et al., 2015; Wright et al., 2007).

*Behavior and Food Preferences*

Nine studies investigated 16 correlates related to child behavior and food preferences (Blossfeld et al., 2007a; Cao et al., 2012; Cassells et al., 2014; Fildes et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Monnery-Patris et al., 2015; Reau et al., 1996; Wright et al., 2007). Picky eating was associated with lower appetite (Wright et al., 2007), enjoyment of food (Fildes et al., 2015), food responsiveness (Cao et al., 2012; Fildes et al., 2015), and desire to drink (Cao et al., 2012). One study found a positive association between differential reactivity to smell stimuli and food neophobia but no association between differential reactivity to taste stimuli and food neophobia (Monnery-Patris et al., 2015). Picky eating was associated with higher satiety responsiveness (Fildes et al., 2015), longer feeding time (Cao et al., 2012; Fildes et al., 2015; Reau et al., 1996), and liking fewer foods (Wright et al., 2007), such as fruits and vegetables (Fletcher et al., 2016; Howard et al., 2012). There was no association between picky eating and emotional overeating or undereating (Cao et al., 2012), temperament (difficultness) (Cassells et

al., 2014), sour taste acceptance (Blossfeld et al., 2007a), or liking of discretionary foods (Howard et al., 2012).

*Dietary Intake*

Eleven studies investigated a total of 16 correlates related to the child's dietary intake (Barends et al., 2014; Blossfeld et al., 2007b; Briefel et al., 2006; Cardona Cano et al., 2015; Carruth et al., 2004a; Fletcher et al., 2016; Howard et al., 2012; Northstone & Emmett, 2013; Perry et al., 2015; Sauve & Geggie, 1991; Wright et al., 2007). One cross-sectional study found that picky eating was associated with consuming a smaller variety of foods regardless of the child's dietary intake pattern (Northstone & Emmett, 2013). Another cross-sectional study found that intake of complex food textures (specifically, chopped carrots) was associated with lower food neophobia, pickiness, and food fussiness (Blossfeld et al., 2007b). In general, studies found that picky eating was associated with trying fewer foods (Cardona Cano et al., 2015; Wright et al., 2007) and having lower intake of foods, such as whole grains (Cardona Cano et al., 2015), rice/pasta (Cardona Cano et al., 2015), meat (Cardona Cano et al., 2015), fish (Cardona Cano et al., 2015), and vegetables (Barends et al., 2014; Cardona Cano et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Perry et al., 2015). Evidence for fruit and discretionary food intake (i.e., salty snacks and sweets) was mixed. Two cross-sectional studies found a negative association between food neophobia and fruit intake (Fletcher et al., 2016; Perry et al., 2015), but 3 studies found no association (Barends et al., 2014; Cardona Cano et al., 2015; Howard et al., 2012). One study found a positive association between food neophobia and discretionary food intake (Perry et al., 2015), whereas another study found a negative association between sweet intake and picky eating but no association with savory snack intake (Cardona Cano et al., 2015). There was no association between picky eating and intake of refined grains, dairy, or composite dishes

(Cardona Cano et al., 2015). Evidence for energy intake was mixed. Two studies found that picky eating was associated with lower caloric intake (Cardona Cano et al., 2015; Carruth et al., 2004a) and 1 longitudinal study found no association (Sauve & Geggie, 1991). One cross-sectional study found that picky eating was associated with lower micronutrient intake (Carruth et al., 2004a). Interestingly, 1 study found that picky eating was positively associated with the child's intake of vitamin and mineral supplements (Briefel et al., 2006).

### *Clan: Parent Characteristics and Family Dynamics*

#### *Demographics*

Four studies investigated demographic correlates in the family (Carruth et al., 2004a; Cassells et al., 2014; Hendricks et al., 2006; Migraine et al., 2013). Maternal education was the most frequently used proxy for socioeconomic status. Null findings were reported in general for the association between socioeconomic status and picky eating; however, one study found that maternal education beyond high school was associated with lower risk of picky eating behavior (Migraine et al., 2013). In terms of maternal age, one longitudinal study found a positive association between maternal age at delivery and child food neophobia (Cassells et al., 2014), and two cross-sectional studies found no association (Hendricks et al., 2006; Migraine et al., 2013). Only one study investigated parent race/ethnicity and found no association with picky eating (Hendricks et al., 2006). There was no association between marital status and picky eating (Carruth et al., 2004a). There was no association between picky eating and the number of children in the household younger than 18 years of age (Hendricks et al., 2006).

#### *Parent Diet and Health*

Four studies investigated characteristics of caregiver's diet and health (Cao et al., 2012; Cassells et al., 2014; Maslin et al., 2015; Migraine et al., 2013). Null findings were reported for

the association between parental weight and picky eating (Cao et al., 2012; Cassells et al., 2014; Migraine et al., 2013). Only 1 study investigated maternal diet, and it found a positive association between child food neophobia and the percentage of fruits and vegetables disliked by the mother (Cassells et al., 2014). There was no association between picky eating and percentage of other foods disliked by the mother (Cassells et al., 2014) or maternal food allergy history (Maslin et al., 2015).

*Early Feeding Characteristics*

Nine studies examined 11 correlates related to feeding characteristics during infancy (Brown & Lee, 2015; Cardona Cano et al., 2015; Carruth et al., 2004a; Cassells et al., 2014; Maslin et al., 2015; Migraine et al., 2013; Northstone et al., 2001; Syrad et al., 2015; Yuan et al., 2016). There were null findings for cross-sectional associations between ever- breastfed status and picky eating (Carruth et al., 2004a; Maslin et al., 2015; Migraine et al., 2013). The evidence for picky eating and breastfeeding duration was mixed. One longitudinal study found a negative association with food fussiness (Brown & Lee, 2015), whereas another longitudinal study found no association with food neophobia (Cassells et al., 2014). There was no association between food neophobia and long breastfeeding or later introduction of main meal food (Yuan et al., 2016), nor between picky eating and formula intake (Cardona Cano et al., 2015) or mode of feeding (Cassells et al., 2014). One longitudinal study found that food fussiness in children at 16 months was positively associated with extended formula feeding at 21 months (Syrad et al., 2015). Picky eating was positively associated with excluding cow's milk for presumed cow's milk allergy (Maslin et al., 2015). One study found that baby-lead weaning (emphasis on infant self-feeding whole foods rather than parent spoon-feeding pureed foods) was negatively associated with picky eating (Brown & Lee, 2015). Three longitudinal studies investigated

child's age when first given solids and picky eating with mixed results (Brown & Lee, 2015; Cassells et al., 2014; Northstone et al., 2001). One study found that introducing complementary foods at an earlier age was positively associated with fussy eating (Brown & Lee, 2015). However, another study found that introducing solids before age 6 months was associated with fewer picky eating behaviors at 15 months, and introducing solids at age 10 months or later was associated with more picky eating behaviors (Northstone et al., 2001). Another study found no association between the age a child was first given solids and food neophobia (Cassells et al., 2014). One longitudinal study found that food neophobia at 1 year was positively associated with later introduction of dairy products and use of ready-prepared baby foods and the use of ready-prepared adult foods (Yuan et al., 2016).

*Feeding Beliefs and Practices*

Five studies investigated 14 correlates related to parental feeding beliefs and practices (Brown & Lee, 2015; Carruth et al., 2004a; Cassells et al., 2014; Jansen et al., 2014; Johnson & Harris, 2004). In general, both cross-sectional and longitudinal studies used continuous measures of picky eating or food neophobia (i.e., CEBQ food fussiness subscale and CFNS). Picky eating was positively associated with mealtime negativity (Johnson & Harris, 2004) and maternal concern about infant undereating and becoming underweight (Brown & Lee, 2015; Cassells et al., 2014). Positive feeding practices, often termed "responsive feeding", are child-centered in nature and facilitate the development of the child's recognition of their hunger and satiety cues (Black & Aboud, 2011). There was an inverse relationship between picky eating and positive feeding practices, specifically maternal awareness of infant hunger and satiety cues (Cassells et al., 2014), monitoring (the extent to which parents oversee their child's eating) (Brown & Lee, 2015), and setting structured family meals (Jansen et al., 2014). Negative feeding practices,

termed “nonresponsive feeding”, are parent-centered in nature and hinder the development of the child’s recognition of their hunger and satiety cues (Black & Aboud, 2011; Jansen et al., 2014). There was a positive relationship between picky eating and negative feeding practices, particularly distrust in the child’s appetite (Jansen et al., 2014), overt restriction (Brown & Lee, 2015; Cassells et al., 2014; Jansen et al., 2014), and rewarding the child for eating or good behavior (Jansen et al., 2014). Two studies found a positive association between pressure to eat/persuasive feeding (Cassells et al., 2014; Jansen et al., 2014), but 1 study found no association (Brown & Lee, 2015). There was no association between picky eating and covert restriction or structured meal timing (Jansen et al., 2014) or how many times caregivers offered a new food before deciding the child disliked it (Carruth et al., 2004a). Overt restriction refers to restriction that the child can see and is aware of, whereas covert restriction refers to restriction that the child cannot see and is not aware of (Jansen et al., 2014).

***Community/Country: Access to Food, Social Influences***

Three studies investigated factors in the community and country level (Carruth et al., 2004a; Hendricks et al., 2006; Magarey et al., 2016). The single RCT included in the review found no association between providing guidance on protective feeding practices and picky eating 6 months later (Magarey et al., 2016). There was no association between picky eating and region of residence (Northwest, Midwest, South, West) (Hendricks et al., 2006) or settlement (urban, suburban, rural) (Carruth et al., 2004a). One cross-sectional study found a negative association between children receiving the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) and picky eating (Hendricks et al., 2006). There was no association between the child being in childcare and picky eating (Hendricks et al., 2006).

**Meta-Analysis of Picky Eating Correlates**

**Figures 2.2** and **2.3** show the forest plots from the meta-analysis. The estimated prevalence of picky eating in children aged 4-30 months from the meta-analysis is 22.0% (95% CI, 11.5%-32.5%; $I^2$, 99.5%; random-effects model) based on 5 studies that reported a dichotomous definition for picky eating (Cardona Cano et al., 2015; Carruth et al., 2004a; Northstone & Emmett, 2013; Reau et al., 1996; Wright et al., 2007). The degree of picky eating estimated in meta-analysis is 2.51 (95% CI, 2.42-2.59; $I^2$, 99.6%; random-effects model) on a scale ranging from 1 (never food fussy) to 5 (always food fussy) based on 6 studies that used the CEBQ food fussiness subscale (Blossfeld et al., 2007b; Brown & Lee, 2015; Fildes et al., 2015; Magarey et al., 2016; Svensson et al., 2011; Syrad et al., 2015). There was no indication of publication bias from either the Begg's test with continuity correction ($P = 0.47$) or the Egger's test ($P = 0.74$). Meta-regression found that among children aged 12-24 months, each additional month older was associated with an increase in the CEBQ food fussiness score by 0.06 units (95% CI, 0.01-0.11; random-effects model).

**Study Quality**

**Table 2.4** reports the overall results of the study quality assessment for the 32 studies included in the review. On average, studies scored 8.09 out of 12 (range, 5-11). The large majority of studies (84%) clearly specified and defined the study population (Barends et al., 2014; Blossfeld et al., 2007a; Blossfeld et al., 2007b; Brown & Lee, 2015; Cao et al., 2012; Cardona Cano et al., 2015; Cassells et al., 2014; Fildes et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Jansen et al., 2014; Johnson & Harris, 2004; Johnson et al., 2016; Magarey et al., 2016; McCarthy et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Northstone & Emmett, 2013; Northstone et al., 2001; Oliveira et al., 2015; Perry et al., 2015; Quah et al., 2015;

Sauve & Geggie, 1991; Svensson et al., 2011; Syrad et al., 2015; Wright et al., 2007; Yuan et al., 2016). However, aims/hypotheses were more variable, and only 13 studies (41%) explicitly stated picky eating or food neophobia in the objective or hypothesis (Blossfeld et al., 2007a; Cardona Cano et al., 2015; Carruth et al., 2004a; Cassells et al., 2014; Fildes et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Johnson & Harris, 2004; Magarey et al., 2016; Maslin et al., 2015; Monnery-Patris et al., 2015; Perry et al., 2015; Yuan et al., 2016). Five studies (16%) were longitudinal with sample sizes of >1000 participants (Cardona Cano et al., 2015; Northstone et al., 2001; Oliveira et al., 2015; Syrad et al., 2015; Yuan et al., 2016). More than half of the studies used reliable and valid measures for both picky eating and other investigated correlates (Blossfeld et al., 2007a; Blossfeld et al., 2007b; Cao et al., 2012; Cardona Cano et al., 2015; Cassells et al., 2014; Fildes et al., 2015; Howard et al., 2012; Jansen et al., 2014; Johnson & Harris, 2004; Johnson et al., 2016; McCarthy et al., 2015; Migraine et al., 2013; Monnery-Patris et al., 2015; Oliveira et al., 2015; Perry et al., 2015; Quah et al., 2015; Svensson et al., 2011).

## 2.8 Discussion

This study systematically reviewed existing evidence on the correlates of picky eating behavior among young children. In the 32 included studies, the most examined correlates were characteristics of the child, specifically the child's sex, current weight, and dietary intake. Characteristics of the parents were also extensively examined, particularly early feeding characteristics and feeding beliefs and practices. These findings are similar to those of previous reviews on picky eating that included older children, in which associations were primarily related to the child's weight status (Brown et al., 2016b), dietary intake (Taylor et al., 2015), and social

influences (Lafraire et al., 2016). However, unlike previous reviews, this study identified associations related to biological contributions and access to food. From meta-analysis, the prevalence of picky eating was 22.0% and the degree was 2.51 (sometimes fussy) on the CEBQ food fussiness subscale. This suggests that almost a quarter of children aged ≤2 years are picky eaters, but overall, parents perceive picky eating as an occasional behavior. There was a statistically significant relationship between age and picky eating; each additional month in age was associated with an increase in the CEBQ food fussiness score by 0.06 U. This indicates that picky eating behavior could increase from rarely to occasionally (or occasionally to frequently) over the course of 1 year in young children. Similar to previous reviews on picky eating that included older children (Brown et al., 2016b; Taylor et al., 2015), there was a wide range in prevalence estimates due to differences in conceptualization and assessment tools. Until a uniform definition of picky eating is agreed upon, future studies should consider providing data from both categorical and continuous measures of picky eating in order assess prevalence and severity. Studies that utilize a continuous measure of picky eating and food neophobia provide information on the degree of picky eating but cannot be used to assess prevalence or compare picky eaters with nonpicky eaters. To account for this, a single question (e.g., "Is your child a picky eater?") can be used in conjunction with continuous measures to assess both the degree and severity of picky eating. If continuous variables are categorized, then researchers should consider categorizing picky eating on an ordinal scale (e.g., no/low picky eating, moderate/sometimes picky eating, high/frequent picky eating) instead of dichotomizing picky eating scores so that the degree of picky eating can be measured more precisely.

The majority of studies (7 of 8) found no association between the child's sex and picky eating, which corresponds with previous research in preschool-aged children (Dubois et al.,

2007a; Russell & Worsley, 2008). However, 1 study of Chinese toddlers found that girls had more food fussiness than boys (Cao et al., 2012). This may be explained by differences in cultural background and feeding practices (Huang et al., 2012), or by the fact that picky eating in this study was reported by the parent or grandparent. Holm-Denoma et al. (2005) found that fathers, compared with mothers, rated their child as pickier; in addition, parents reported eating behaviors differently depending on the child's sex. Future research on picky eating should account for sex differences of both the child and the caregiver. Although there was no association between birth weight and picky eating, the evidence for current weight was mixed. These results are similar to findings from a recent systematic review that included older children and found no association between childhood weight status and picky eating, which is likely due to discrepancies in the definition and measurement of picky eating (Brown et al., 2016b). Several studies investigated dietary intake and found a negative association with picky eating. Not surprisingly, the strongest evidence was an association with lower vegetable intake (Barends et al., 2014; Cardona Cano et al., 2015; Fletcher et al., 2016; Howard et al., 2012; Perry et al., 2015). One concern regarding picky eating is the intake of sweets and savory snacks; in this regard, the present review had mixed results, similar to findings in older children (Jacobi et al., 2003; van der Horst et al., 2016). Interestingly, picky eating was positively associated with the child's vitamin and supplement intake (Briefel et al., 2006). However, a recent study found that, other than an association with lower iron and zinc intake, picky eating does not compromise nutrient intake (Taylor et al., 2016). This may provide reassurance to some parents, who could be adding vitamin and mineral supplements to their child's diet in order to compensate for picky eating behavior (Fox et al., 2006).

Numerous correlates related to caregiver feeding were investigated. In terms of early feeding characteristics, evidence was inconsistent for the association between breastfeeding duration and picky eating, and null findings were reported for ever-breastfed status. This differs from previous findings in older children, which have indicated an inverse association between being ever breastfed and picky eating (Shim et al., 2011; van der Horst et al., 2016). Farrow and Blissett (2006) found that breastfeeding, when mediated by lower reported maternal control over child feeding, predicted less negative mealtime interactions between mothers and infants at 1 year of age. However, there was no association between breastfeeding and observed infant acceptance/rejection of food. Further research is needed to explore the relationship between breastfeeding and picky eating behavior. Previous studies suggested that picky eating was associated with early introduction of complementary foods (Shim et al., 2011). In the present review, results were inconsistent for the relationship between the age at which a child was first given solids and picky eating.

Along the lines of complementary feeding, 1 study found that a baby-led approach was negatively associated with picky eating (Brown & Lee, 2015). The World Health Organization currently recommends that infants begin complementary foods at 6 months of age (World Health Organization (WHO), 2004, 2005). However, research is sparse regarding how to introduce complementary foods and the use of baby-led weaning as an alternative to standard approaches (Daniels et al., 2015). In a recent review of qualitative studies on parental perception of healthy behaviors in preventing overweight in children, picky eating was identified as a barrier to healthy eating (Pocock et al., 2010). Parents of picky eaters are more likely be concerned about undereating (Brown et al., 2016a), which could lead to the use of both positive and negative feeding practices in an attempt to influence their children's food preferences and dietary intake

(Russell et al., 2015). Responsive feeding involves a division of responsibility between the parent and child during mealtimes—parents are responsible for what, when, and where the child eats, whereas the child is responsible for how much and whether or not to eat (Satter, 1986). This review identified a negative relationship between picky eating and responsive feeding such as an increased awareness of infant hunger and satiety cues, monitoring, and providing structured family meals (Brown & Lee, 2015; Cassells et al., 2014; Jansen et al., 2014). In contrast, nonresponsive feeding practices (such as distrust in the child's appetite, overt restriction, pressure to eat, and rewards for eating or good behavior) were positively associated with picky eating behavior (Brown & Lee, 2015; Cassells et al., 2014; Jansen et al., 2014). These findings suggest that responsive feeding may play a role in the development of healthy independent eating and highlights the importance of reciprocity in feeding interactions. Additionally, based on a systematic review on responsive feeding and overweight in infants and toddlers, further research is needed in this area for early childhood obesity prevention (DiSantis et al., 2011).

Additional research is needed at the cell and community/country levels. Twin studies in older children have demonstrated that picky eating and food neophobia are highly heritable traits, with estimates ranging 72%-78% (Cooke et al., 2007; Faith et al., 2013; Fildes et al., 2016). Previous research in preschool-aged children suggests that genetic sensitivity to bitter taste may influence the development of picky eating behavior (Keller et al., 2002; Tsuji et al., 2012); however, similar studies in children aged ≤ 2 years were not identified. The present review showed that picky eating behavior was positively associated with preterm birth, which supports previous results indicating that preterm birth may play a role in infant feeding problems (Crapnell et al., 2013; Silberstein et al., 2009). However, other factors could lead to feeding problems in young children, such as food allergies, gastro-esophageal reflux disease, and

frequent respiratory infections (Field et al., 2003). Further research is needed to investigate additional genetic and biological factors related to picky eating in young children and to determine their influence on picky eating in comparison to other problematic feeding disorders.

At the community/country level, additional areas for research include WIC participation and childcare, specifically the influence of childcare providers. Similar to another cross-sectional study in older children (Evans et al., 2011), an inverse association between picky eating and WIC participation was found. WIC nutrition staff often receive training on strategies parents can use to manage picky eating behavior (Colorado Department of Public Health & Environment; Washington State WIC Nutrition Program, 2011); thus WIC participants may receive more guidance on picky eating. Another possibility is that high-income non-WIC participants may cater to their child's mealtime requests differently (Evans et al., 2011). Millions of young American children spend time in childcare (Laughlin, 2013), and further study of the role of childcare could provide valuable information to address the development of picky eating behavior in children. Both the Academy of Nutrition and Dietetics and the Institute of Medicine recommend that childcare providers practice the division of responsibility and responsive feeding (Benjamin Neelon et al., 2011; Institute of Medicine, 2011). A recent study on infant and toddler feeding practices in licensed childcare centers found that some providers use nonresponsive feeding practices during family-style meals, such as trying to get children to finish the food they had self-served (Blaine et al., 2015). Another study found that childcare providers across different contexts (i.e., Head Start, Child and Adult Care Food Program, and non-Child and Adult Care Food Program) did not meet the Academy of Nutrition and Dietetic's recommendation to help children understand feelings of hunger and satiety (Dev et al., 2013). Additional research is needed to investigate the impact of nonparental care (e.g., feeding styles

and practices) on dietary intake and eating behavior among infants and toddlers, and to compare the perception of picky eating behavior between childcare providers and parents.

Findings of this review could inform the Birth to 24-months and Pregnant Women Dietary Guidance Development Project (B-24 Project), a joint effort led by the U.S. Department of Agriculture and the U.S. Department of Health and Human Services to evaluate the evidence base to support the inclusion of children from birth to 24 months of age in the Dietary Guidelines for Americans (DGA) (Raiten et al., 2014). The current DGA provides food-based recommendations for adults and children aged ≥ 2 years (U.S. Department of Health and Human Services [DHHS] and U.S. Department of Agriculture [USDA], 2015), and currently there are no guidelines similar to the DGA for children aged ≤2 years. One task of the B-24 Project is to identify the micro-environmental effects on the transition to an adult diet during the 12-24 month period, including factors related to picky eating. By identifying correlates of picky eating from birth to 2 years, this review helps inform the development of evidence-based guidance and recommendations that promote optimal nutrition and weight-gain trajectories in young children. Additionally, this review identifies areas related to parent and caregiver feeding practices where additional research is warranted.

Some limitations need to be considered when interpreting the findings of this review. There is no consensus on the operational definition of picky eating and no standardized instrument to assess corresponding behaviors in children. This review included studies that measured picky eating and food neophobia using a variety of tools; however, all picky eating assessments were based on caregiver perception. Although many studies examined ≥2 correlates of picky eating behavior, few studies used the same measurement tool, which limited the ability to conduct additional extensive meta-analyses and investigate the impact of different covariates

on picky eating. This review is limited to peer-reviewed articles in English; thus, research published in other languages and unpublished studies were excluded. Studies that examined food refusal or struggles for control alone without linking to picky eating or food neophobia were also excluded. Additionally, literature search, full-text review, and data extraction were conducted by a single author.

## 2.9 Conclusion

Picky eating among children is a common concern, yet little is known about how these behaviors develop in early childhood. Without an operational definition and standard measurement tool, the prevalence of picky eating behavior and its relationship with other correlates will continue to show high variability. Future studies should provide data from both categorical and continuous measures to quantify the degree and severity of picky eating. The present systematic review found that influences on children's picky eating behavior are present at each examined level, from cell to society, but they are predominantly centered around the child and the parent(s). This highlights the importance of investigating the family unit when conducting picky eating research, particularly in regards to child-parent dyads and bidirectional feeding interactions. By using a socio-ecological model to characterize the evidence on picky eating, this review identifies modifiable factors that may promote healthy dietary habits and food preferences in the first 2 years of life and draws attention to the lack of research at the level of the cell and community/country.

## 2.10 Tables and Figures

**Table 2.1.** Summary of genetic variants potentially related to picky eating

| Gene | Polymorphism | Major/Minor Alleles | MAF[a] | Related Phenotype |
|---|---|---|---|---|
| *BDNF* | rs6265 | G/A | 0.20 | Appetitive Traits |
| *CA6* | rs2274327 | C/T | 0.27 | Taste Perception |
| | rs2274333 | A/G | 0.34 | |
| *COMT* | rs4680 (Val$^{158}$Met) | G/A | 0.37 | Temperament |
| *DAT1* | rs40184 | 0.41 | 0.41 | Temperament |
| *DRD2* | rs1800497 (Taq1A) | C/T | 0.33 | Temperament |
| | rs2283265 | G/A | 0.23 | |
| *DRD4* | rs1800955 | T/C | 0.41 | Temperament |
| *FTO* | rs8057044 | A/G | 0.46 | Appetitive Traits |
| *GHRL* | rs27647 | T/C | 0.21 | Appetitive Traits |
| | rs696217 | G/T | 0.09 | |
| *GNAT3* | rs7792845 | C/T | 0.32 | Taste Perception |
| *LEPR* | rs1137101 | G/A | 0.48 | Appetitive Traits |
| *LEP* | rs7799039 | G/A | 0.32 | Appetitive Traits |
| | rs11761556 | C/A | 0.46 | |
| | rs1349419 | A/G | 0.45 | |
| *MC4R* | rs17782313 | T/C | 0.26 | Appetitive Traits |
| *PYY* | rs2070592 | A/G | 0.45 | Appetitive Traits |
| | rs2014257 | T/A | 0.27 | |
| | rs228771 | C/G | 0.19 | |
| *TAS1R1* | rs34160967 | C/A | 0.14 | Taste Perception |
| *TAS1R2* | rs35874116 | T/C | 0.27 | Taste Perception |
| *TAS1R3* | rs307355 | C/T | 0.24 | Taste Perception |
| | rs35744813 | G/A | 0.28 | |
| *TAS2R1* | rs2234233 | C/A | 0.11 | Taste Perception |
| *TAS2R19* | rs10772420 | G/A | 0.35 | Taste Perception |
| *TAS2R38* | rs713598 | G/C | 0.49 | Taste Perception |
| | rs1726866 | C/T | 0.43 | |
| | rs10246939 | T/C | 0.48 | |

[a] Caucasian population

**Table 2.2.** Criteria used to define the research question for the systematic review

| Criteria | Description |
|---|---|
| Population | Children aged ≤ 30 mo without chronic diseases, developmental disability, autism spectrum disorder, or avoidant/restrictive food intake disorder |
| Intervention | Any cross-sectional or experimental study examining a correlate of picky eating or food neophobia |
| Comparison | Not applicable |
| Outcome(s) | Picky eating, pickiness, fussy eating, choosy eating, faddy eating, food neophobia, food fussiness, or selective eating |

**Table 2.3.** Basic characteristics of the studies on the correlates of picky eating and food neophobia among young children

| Reference | Country | Study design | Sample size | Age (mo) | Measure of PE or FN | Method of reporting PE or FN | Prevalence of PE or FN | Specific behavior of interest |
|---|---|---|---|---|---|---|---|---|
| (Barends et al., 2014) | Netherlands | Longitudinal | 81 | 12-23 | Single questions | Maternal | NR | Picky eating, low food neophobia[a] |
| (Blossfeld et al., 2007a) | Ireland | Cross-sectional | 53 | 18 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness |
| (Blossfeld et al., 2007b) | Ireland | Cross-sectional | 70 | 12 | Picky eating questionnaire; Children's Eating Behavior Questionnaire | Maternal | NR | Picky eating, low food neophobia[a]; food fussiness |
| (Briefel et al., 2006) | USA | Cross-sectional | 3022 | 4-24 | Single question | Parent or primary caregiver | NR | Picky eating |
| (Brown & Lee, 2015) | UK | Longitudinal | 298 | 18-24 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness |
| (Cao et al., 2012) | China | Cross-sectional | 219 | 12-18 | Children's Eating Behavior Questionnaire | Parent or grandparent | NR | Food fussiness |
| (Cardona Cano et al., 2015) | Netherlands | Longitudinal | 3618 | 18 | Child Behavior Checklist (two items) | Maternal | 26.5% | Picky eating |
| (Carruth et al., 2004a) | USA | Cross-sectional | 3022 | 4-24 | Single question | Parent or primary caregiver | 30.5% | Picky eating |
| (Cassells et al., 2014) | Australia | Longitudinal | 244 | 24 | Child Food Neophobia Scale | Maternal | NR | Food neophobia |
| (Fildes et al., 2015) | UK | Cross-sectional | 1044 | 16 | Children's Eating Behavior Questionnaire | Parent | NR | Food fussiness |

**Table 2.3. (cont.)**

| Reference | Country | Study design | Sample size | Age (mo) | Measure of PE or FN | Method of reporting PE or FN | Prevalence of PE or FN | Specific behavior of interest |
|---|---|---|---|---|---|---|---|---|
| (Fletcher et al., 2016) | UK | Cross-sectional | 456 | 30 | Single question | Maternal | NR | Food neophobia |
| (Hendricks et al., 2006) | USA | Cross-sectional | 2515 | 4-24 | Single question | Parent or primary caregiver | 42.2%[b] | Picky eating |
| (Howard et al., 2012) | Australia | Cross-sectional | 245 | 24 | Child Food Neophobia Scale | Maternal | NR | Food neophobia |
| (Jansen et al., 2014) | Australia | Cross-sectional | 462 | 21-27 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness |
| (Johnson & Harris, 2004) | UK | Cross-sectional | 18 | 15-21 | CCTI Reaction to Food scale | Maternal | NR | Food neophobia |
| (Johnson et al., 2016) | UK | Cross-sectional | 1387 | 24[c] | Eating behavior questionnaire | Parent | NR | Picky eating/refusal |
| (Magarey et al., 2016) | Australia | RCT (midline data) | 698 | 24 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness |
| (Maslin et al., 2015) | UK | Cross-sectional | 126 | 8-30 | Picky eating questionnaire | Maternal | NR | Fussy eating |
| (McCarthy et al., 2015) | Ireland | Cross-sectional | 1189 | 24 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness |
| (Migraine et al., 2013) | France | Cross-sectional | 479 | 24[c] | Children's Eating Difficulties Questionnaire | Maternal | NR | Low food variety (picky eating + food neophobia) |
| (Monnery-Patris et al., 2015) | France | Cross-sectional | 123 | 20-22 | Children's Eating Difficulties Questionnaire | Maternal | NR | Food neophobia |

**Table 2.3. (cont.)**

| Reference | Country | Study design | Sample size | Age (mo) | Measure of PE or FN | Method of reporting PE or FN | Prevalence of PE or FN | Specific behavior of interest |
|---|---|---|---|---|---|---|---|---|
| (Northstone & Emmett, 2013) | UK | Cross-sectional | 9599 | 24 | Single question | Maternal | 9.8% | Choosy (picky) eating |
| (Northstone et al., 2001) | UK | Longitudinal | 9360 | 15 | Single question | Maternal | NR | Choosy (picky) eating |
| (Oliveira et al., 2015) | France | Longitudinal | 1077 | 24 | Study-specific questions | Maternal | NR | Food neophobia |
| (Perry et al., 2015) | Australia | Cross-sectional | 330 | 24 | Child Food Neophobia Scale | Maternal | NR | Food neophobia |
| (Quah et al., 2015) | Singapore | Longitudinal | 210 | 12-24 | Children's Eating Behavior Questionnaire | Maternal | NR | Food fussiness/enjoyment of food |
| (Reau et al., 1996) | USA | Cross-sectional | 151 | 13-27 | Single question | Parent | 36% | Picky eating |
| (Sauve & Geggie, 1991) | Canada | Longitudinal | 232 | 4-24[c] | Parent interview[d] | Parent | 7.3%[e] | Fussy eating |
| (Svensson et al., 2011) | Sweden | Longitudinal | 55 | 12-24 | Children's Eating Behavior Questionnaire | Parent | NR | Food fussiness |
| (Syrad et al., 2015) | UK | Longitudinal | 1897 | 16 | Children's Eating Behavior Questionnaire | Parent | NR | Food fussiness |
| (Wright et al., 2007) | UK | Cross-sectional | 455 | 30 | Single question | Parent | 8.2% | Faddy (picky) eating |
| (Yuan et al., 2016) | France | Longitudinal | 1142 | 12-24 | Study-specific questions | Maternal | NR | Food neophobia |

**Table 2.3. (cont.)**

*Abbreviations*: CCTI, Colorado Childhood Temperament Inventory; FN, food neophobia; NR, not reported; PE, picky eating; RCT, randomized controlled trial

[a] Study measured openness/willingness to new foods (low food neophobia)

[b] Based on data from the same cohort as (Carruth et al., 2004a), but using a smaller sample size. Thus, only (Carruth et al., 2004a) was included in the meta-analysis to avoid duplication.

[c] Corrected age (study included children who were born pre-term)

[d] Feeding problem was considered as present only if it was a major concern to parents or if it led to therapy or investigation.

[e] Not based on frequency response or dichotomization, thus not included in meta-analysis.

**Table 2.4.** Correlates of picky eating and food neophobia in young children

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| **Cell: genetic and biological contributions** | | | |
| *In utero factors* | | | |
| Born preterm | (Johnson et al., 2016) | | (Migraine et al., 2013) (PFN) |
| Gestational age | | | (Migraine et al., 2013) (PFN) |
| *Medical issues* | | | |
| Child has an allergy | | | (Hendricks et al., 2006) |
| Child has a long-term medical problem | | | (Hendricks et al., 2006) |
| Illness (diarrhea, constipation, vomiting) | (Johnson & Harris, 2004) (FN) | | |
| Oral-motor-skill function | | | (Johnson & Harris, 2004) (FN) |
| **Child: characteristics of the child** | | | |
| *Demographics* | | | |
| Age | (Barends et al., 2014) (PFN); (Carruth et al., 2004a); (Svensson et al., 2011) | | (Cassells et al., 2014) (FN); (Maslin et al., 2015) |
| Birth order | | | (Carruth et al., 2004a)[b] |
| Sex | (Cao et al., 2012) (girls) | | (Cardona Cano et al., 2015); (Carruth et al., 2004a)[b]; (Cassells et al., 2014) (FN); (Johnson et al., 2016); (Maslin et al., 2015); (Migraine et al., 2013) (PFN); (Monnery-Patris et al., 2015) (FN) |
| Race/ethnicity | | | (Carruth et al., 2004a)[b] |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| *Anthropometrics* | | | |
| Birth weight | | | (Cassells et al., 2014) (FN); (Hendricks et al., 2006); (Migraine et al., 2013) (PFN);(Oliveira et al., 2015) (FN) |
| Current weight | | (Brown & Lee, 2015); (Carruth et al., 2004a); (McCarthy et al., 2015) | (Cao et al., 2012); (Cassells et al., 2014) (FN); (Perry et al., 2015) (FN); (Quah et al., 2015); (Sauve & Geggie, 1991); (Wright et al., 2007) |
| Height | | | (Wright et al., 2007) |
| Weight gain | | | (Quah et al., 2015); (Wright et al., 2007) |
| *Behavior and food preferences* | | | |
| Smell differential reactivity | (Monnery-Patris et al., 2015) (FN) | | |
| Taste differential reactivity | | | (Monnery-Patris et al., 2015) (FN) |
| Appetite | | (Wright et al., 2007) | |
| Enjoyment of food | | (Fildes et al., 2015) | |
| Food responsiveness | | (Cao et al., 2012); (Fildes et al., 2015) | |
| Emotional overeating | | | (Cao et al., 2012) |
| Desire to drink | | (Cao et al., 2012) | |
| Satiety responsiveness | (Fildes et al., 2015) | | |
| Slowness in eating/feeding time | (Cao et al., 2012); (Fildes et al., 2015); (Reau et al., 1996) | | |
| Emotional undereating | | | (Cao et al., 2012) |
| Temperament (difficultness) | | | (Cassells et al., 2014) (FN) |
| Sour taste acceptance | | | (Blossfeld et al., 2007a) |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| Number of foods liked | | (Wright et al., 2007) | |
| Fruit liking | | (Fletcher et al., 2016) (FN); (Howard et al., 2012) (FN) | |
| Vegetable liking | | (Fletcher et al., 2016) (FN); (Howard et al., 2012) (FN) | |
| Discretionary foods liking | | | (Howard et al., 2012) (FN) |
| *Dietary intake* | | | |
| Dietary intake patterns | | (Northstone & Emmett, 2013) | |
| Number of foods tried | | (Cardona Cano et al., 2015); (Wright et al., 2007) | |
| Energy/caloric intake | | (Cardona Cano et al., 2015); (Carruth et al., 2004a) | (Sauve & Geggie, 1991) |
| Micronutrient intake | | (Carruth et al., 2004a) | |
| Vitamin/mineral supplement use | (Briefel et al., 2006) | | |
| Complex texture intake | | (Blossfeld et al., 2007b) (PFN) | |
| Refined grain intake | | | (Cardona Cano et al., 2015) |
| Whole grain intake | | (Cardona Cano et al., 2015) | |
| Rice/pasta intake | | (Cardona Cano et al., 2015) | |
| Dairy intake | | | (Cardona Cano et al., 2015) |
| Fruit intake | | (Fletcher et al., 2016) (FN); (Perry et al., 2015) (FN) | (Barends et al., 2014) (PFN); (Cardona Cano et al., 2015); (Howard et al., 2012) (FN) |
| Vegetable intake | | (Barends et al., 2014) (PFN); (Cardona Cano et al., 2015); (Fletcher et al., 2016) (FN); (Howard et al., 2012) (FN); (Perry et al., 2015) (FN) | |
| Meat intake | | (Cardona Cano et al., 2015) | |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| Fish intake | | (Cardona Cano et al., 2015) | |
| Composite dishes intake | | | (Cardona Cano et al., 2015) |
| Discretionary foods intake | (Perry et al., 2015) (FN) | (Cardona Cano et al., 2015) (sweets) | (Cardona Cano et al., 2015) (savory); (Howard et al., 2012) (FN) |
| **Clan: parent characteristics and family dynamics** | | | |
| *Demographics* | | | |
| Maternal age | (Cassells et al., 2014) (FN) | | (Hendricks et al., 2006); (Migraine et al., 2013) (PFN) |
| Maternal race/ethnicity | | | (Hendricks et al., 2006) |
| Number of children in household < 18 years | | | (Hendricks et al., 2006) |
| Marital status | | | (Carruth et al., 2004a)[b] |
| Employment status/mother currently working | | | (Carruth et al., 2004a)[b] |
| Maternal education | | (Migraine et al., 2013) (PFN) | (Carruth et al., 2004a)[b]; (Cassells et al., 2014) (FN) |
| Household income | | | (Carruth et al., 2004a)[b] |
| *Diet and health* | | | |
| % total fruits disliked by mother | (Cassells et al., 2014) (FN) | | |
| % total vegetables disliked by mother | (Cassells et al., 2014) (FN) | | |
| % total other foods disliked by mother | | | (Cassells et al., 2014) (FN) |
| Having an overweight or obese parent | | | (Cao et al., 2012) |
| Maternal weight | | | (Cassells et al., 2014) (FN); (Migraine et al., 2013) (PFN) |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| Maternal food allergy history | | | (Maslin et al., 2015) |
| *Early feeding characteristics* | | | |
| Ever breastfed | | | (Carruth et al., 2004a)[b]; (Maslin et al., 2015); (Migraine et al., 2013) (PFN) |
| Breastfeeding duration | | (Brown & Lee, 2015) | (Cassells et al., 2014) (FN) |
| Mode of feeding (BF, FF, Both) | | | (Cassells et al., 2014) (FN) |
| Formula intake | | | (Cardona Cano et al., 2015) |
| Extended formula feeding | (Syrad et al., 2015) | | |
| Cow's milk exclusion (for allergy) | (Maslin et al., 2015) | | |
| Age first given solids | (Northstone et al., 2001) (>10 mo) | (Brown & Lee, 2015); (Northstone et al., 2001) (<6 mo) | (Cassells et al., 2014) (FN) |
| Baby-led weaning | | Brown & Lee (2015)(Brown & Lee, 2015) | |
| Long breastfeeding, later main meal food introduction, and use of home-made foods | | | (Yuan et al., 2016) (FN) |
| Later introduction of dairy products and use of ready-prepared baby foods | (Yuan et al., 2016) (FN) | | |
| Use of ready-prepared adult foods | (Yuan et al., 2016) (FN) | | |
| *Feeding beliefs and practices* | | | |
| Offers new food before deciding child dislikes it | | | (Carruth et al., 2004a) |
| Covert restriction | | | (Jansen et al., 2014) |
| Mealtime negativity | (Johnson & Harris, 2004) (FN) | | |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| Concern about infant undereating/underweight | (Brown & Lee, 2015); (Cassells et al., 2014) (FN) | | |
| *Positive practices/responsive feeding* | | | |
| Awareness of infant hunger/satiety cues | | (Cassells et al., 2014) (FN) | |
| Monitoring | | (Brown & Lee, 2015) | |
| Structured meal setting | | (Jansen et al., 2014) | |
| Structured meal timing | | | (Jansen et al., 2014) |
| Family meal setting | | (Jansen et al., 2014) | |
| *Negative practices/nonresponsive feeding* | | | |
| Distrust in child's appetite | (Jansen et al., 2014) | | |
| Overt restriction | (Brown & Lee, 2015); (Cassells et al., 2014) (FN); (Jansen et al., 2014) | | |
| Pressure to eat/persuasive feeding | (Cassells et al., 2014) (FN); (Jansen et al., 2014) | | (Brown & Lee, 2015) |
| Reward for eating | (Jansen et al., 2014) | | |
| Reward for behavior | (Jansen et al., 2014) | | |

**Table 2.4. (cont.)**

| Correlate | Association with PE or FN[a] | | |
|---|---|---|---|
| | **Positive** | **Negative** | **None** |
| **Community/Country: access to food/social influences** | | | |
| Guidance on protective feeding practices | | | (Magarey et al., 2016) |
| Region of residence (Northeast, Midwest, South, West) | | | (Hendricks et al., 2006) |
| Residence (urban, suburban, rural) | | | (Carruth et al., 2004a)[b] |
| Child is a WIC recipient | | (Hendricks et al., 2006) | |
| Child is in childcare | | | (Hendricks et al., 2006) |

*Abbreviations*: FN, food neophobia; PE, picky eating; PFN, both picky eating and food neophobia; WIC, Women, Infants and Children (Special Supplemental Nutrition Program)

[a] Associations are for picky eating unless otherwise specified.

[b] Similar finding in (Hendricks et al., 2006), which was based on the same cohort. To avoid duplication, results are listed only for (Carruth et al., 2004a), which had more complete data.

**Table 2.5.** Study quality assessment

| Criterion of study quality[a] | Mean (SD) |
|---|---|
| 1. A priori aim/hypothesis | 1.28 (0.68) |
| 2. Study population clearly specified and defined | 1.84 (0.37) |
| 3. Study design | 0.41 (0.56) |
| 4. Sample size | 1.22 (0.71) |
| 5. Measure of picky eating is reliable and valid | 1.66 (0.55) |
| 6. Measures of correlates are well-validated | 1.69 (0.47) |
| Total study quality score by summing items 1-6 | 8.09 (1.47) |

[a] Scores for each criterion range 0-2, depending on whether the criterion was unmentioned or unmet (0), partially met (1), or completely met (2). The total score for all 6 criteria for each study was in the range of 0-12.

**Table 2.6.** PRISMA checklist

| Section/topic | # | Checklist item | Reported on page # |
|---|---|---|---|
| Title | 1 | Identify the report as a systematic review, meta-analysis, or both. | 1 |
| Structured summary | 2 | Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number. | 2 |
| Rationale | 3 | Describe the rationale for the review in the context of what is already known. | 3-4 |
| Objectives | 4 | Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS). | 4 |
| Protocol and registration | 5 | Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number. | NA |
| Eligibility criteria | 6 | Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale. | 5 |
| Information sources | 7 | Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched. | 6 |
| Search | 8 | Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated. | 6 |
| Study selection | 9 | State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis). | 7 |
| Data collection process | 10 | Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators. | 7 |
| Data items | 11 | List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made. | 34 |
| Risk of bias in individual studies | 12 | Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis. | 8 |

**Table 2.6. (cont.)**

| Section/topic | # | Checklist item | Reported on page # |
|---|---|---|---|
| Summary measures | 13 | State the principal summary measures (e.g., risk ratio, difference in means). | 8 |
| Synthesis of results | 14 | Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., $I^2$) for each meta-analysis. | 17 |
| Risk of bias across studies | 15 | Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies). | 17 |
| Additional analyses | 16 | Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified. | NA |
| Study selection | 17 | Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram. | 9 |
| Study characteristics | 18 | For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations. | 9-10 |
| Risk of bias within studies | 19 | Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12). | 17 |
| Results of individual studies | 20 | For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot. | 11-16 |
| Synthesis of results | 21 | Present results of each meta-analysis done, including confidence intervals and measures of consistency. | 17 |
| Risk of bias across studies | 22 | Present results of any assessment of risk of bias across studies (see Item 15). | NA |
| Additional analysis | 23 | Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]). | NA |
| Summary of evidence | 24 | Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers). | 18-23 |
| Limitations | 25 | Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias). | 23 |
| Conclusions | 26 | Provide a general interpretation of results in the context of other evidence, and implications for future research. | 24 |

**Table 2.6. (cont.)**

| Section/topic | # | Checklist item | Reported on page # |
|---|---|---|---|
| Funding | 27 | Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review. | 25 |

*From:* Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

**Figure 2.1. A.** Gene-environment interaction and diathesis stress: children who are genetically susceptible to environmental influences (purple dotted line) display more negative outcomes (picky eating) compared to children who are not genetically susceptible (orange solid line). **B.** Gene-environment interaction and differential susceptibility: children who are genetically susceptible to environmental influences (orchids, purple dotted line) display more positive outcomes (non-picky eating) in favorable environments (high feeding responsiveness), and more negative outcomes (picky eating) in unfavorable environments (low feeding responsiveness). These results are more pronounced compared to children who are not genetically susceptible to environmental influences (dandelion, orange solid line).

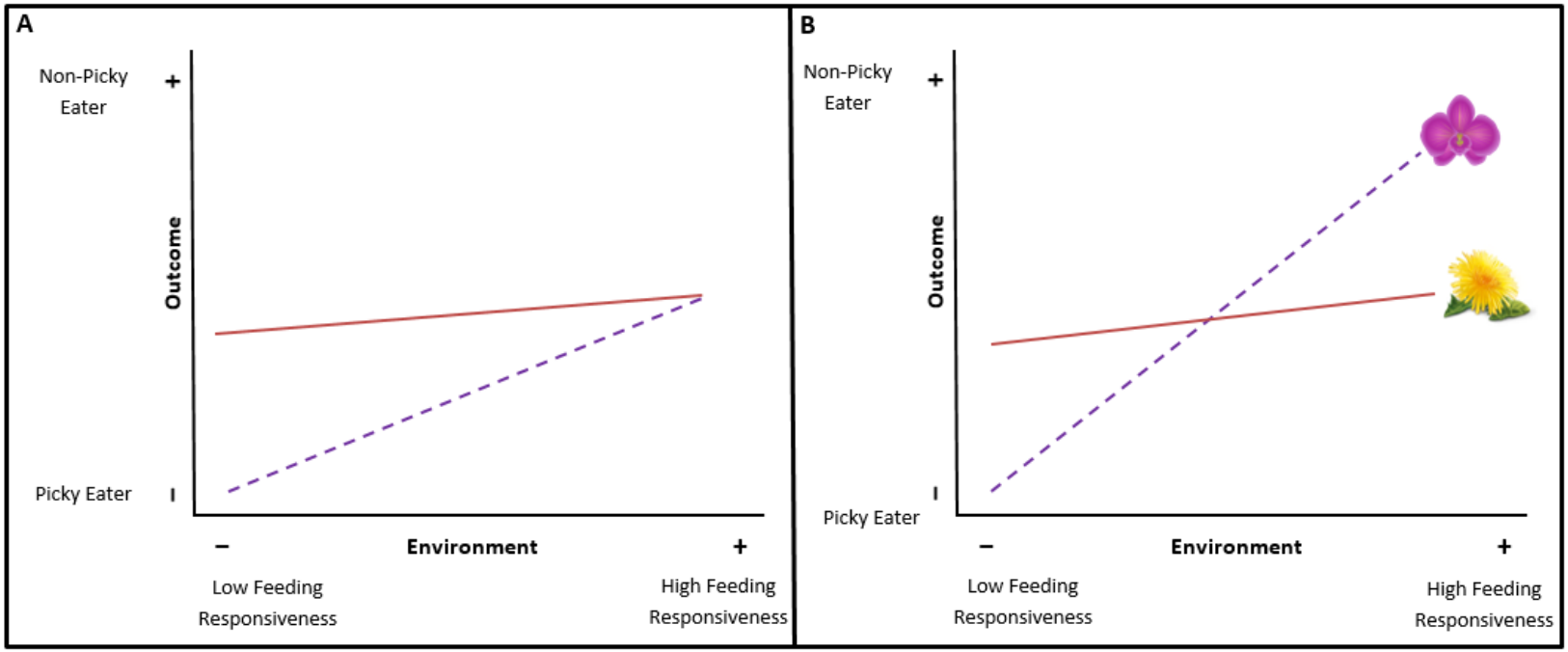


**Figure 2.2.** Study selection flowchart.

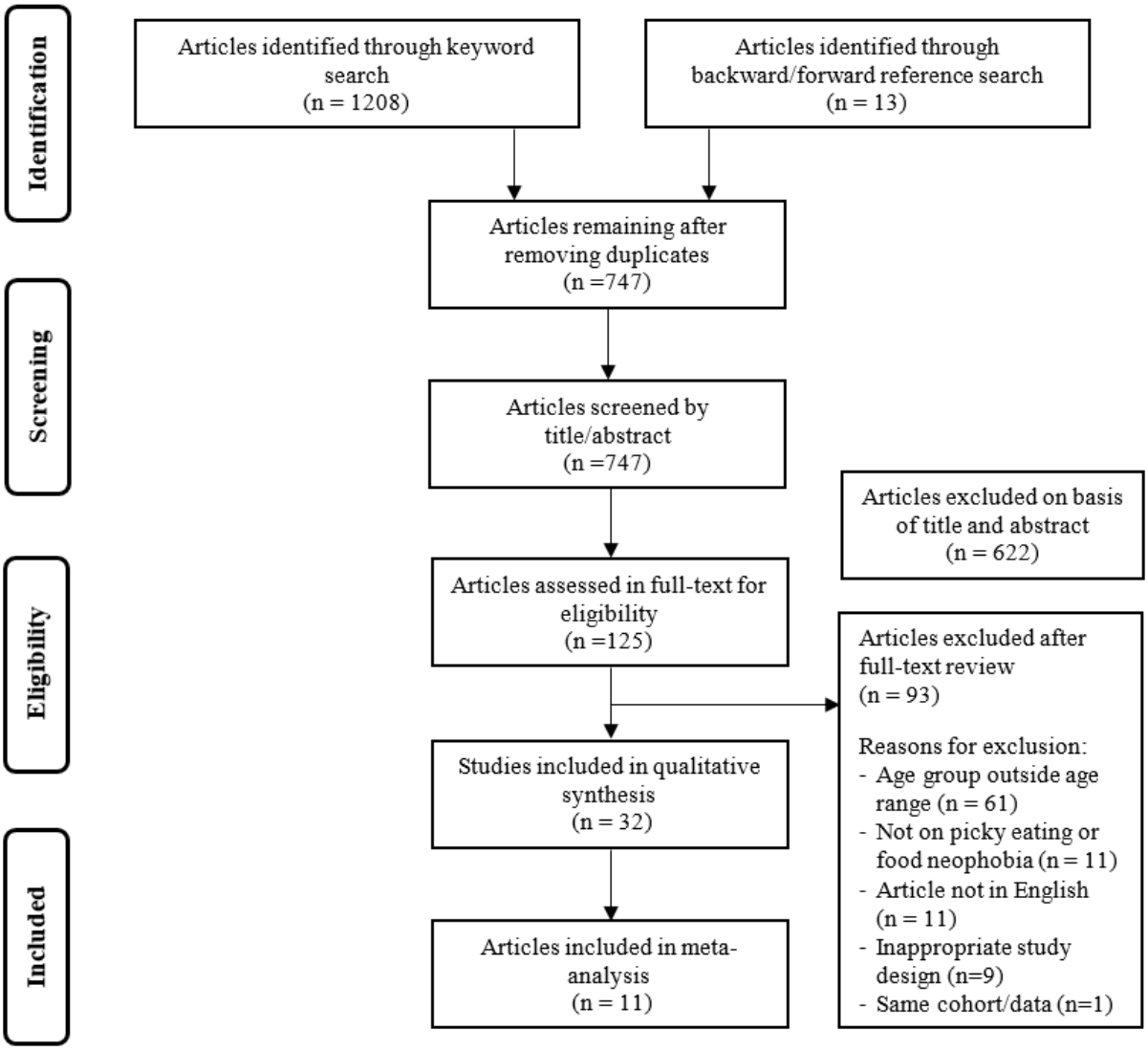


**Figure 2.3.** Forest plot of the prevalence of picky eating from meta-analysis (random effects model). Based on a dichotomous definition for picky eating, the estimated prevalence of picky eating in children aged 4-30 months was 22.0%. Final row indicates overall effect size estimate. *Abbreviation*: CI, confidence interval

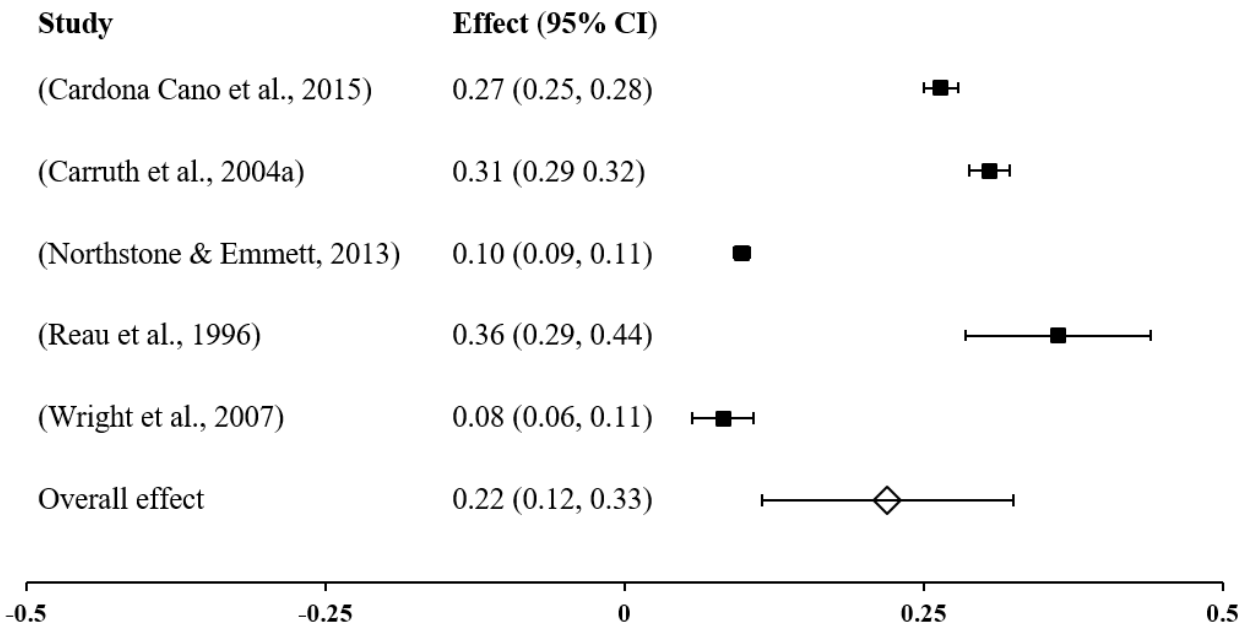
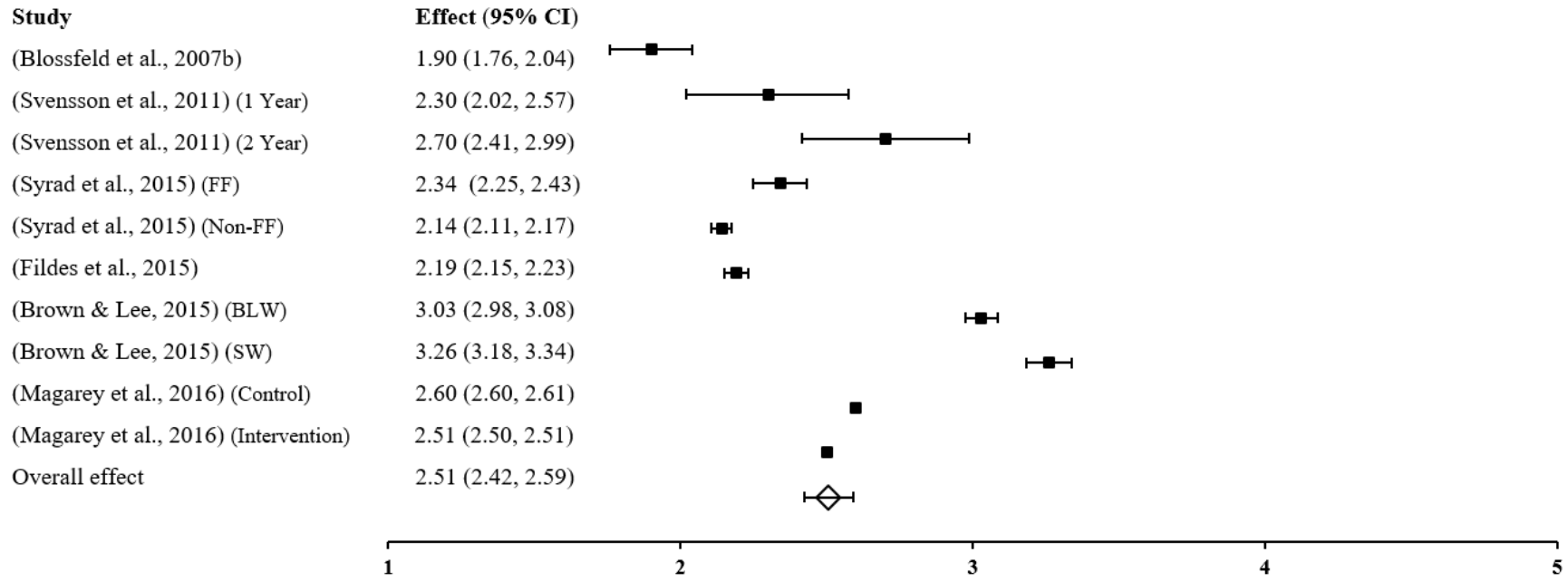


**Figure 2.4.** Forest plot of the degree of picky eating from meta-analysis (random effects model). Based on the Children's Eating Behavior Questionnaire, the degree of food fussiness was 2.51 on a scale of 1-5 (1, never; 5, always). Final row indicates overall effect size estimate. *Abbreviations*: BLW, baby-led weaning group; CEBQ, Child Eating Behavior Questionnaire; CI, confidence interval; FF, formula feeding; SW, standard weaning group.



# CHAPTER 3:
# Variants in Chemosensory Genes are Associated with Picky Eating Behavior in Preschool-Age Children[3]


## Abstract

**Background/Aims:** Picky eating is prevalent among preschoolers and is associated with risk of both underweight and overweight. Although differences in taste perception may be due to genetic variation, it is unclear whether these variations are related to picky eating behavior. The aim of this study was to investigate the association of six single nucleotide polymorphisms (SNPs) in five candidate genes related to chemosensory perception with picky eating behavior and adiposity in a cohort of preschool-aged children.

**Methods:** Parents of 2-5 year-old non-Hispanic white preschoolers (n = 153) responded to survey questions on demographics, and information regarding their child's breastfeeding history and picky eating behavior. Height and weight were measured to calculate body mass index (BMI) *z*-scores using standard growth charts, and saliva was collected for genotyping. Generalized linear models were used to examine associations between picky eating behavior and BMI *z*-scores with genetic variation.

**Results:** When controlling for child age, sex, breastfed status, and parent education level, SNPs in *TAS2R38* (rs713598) and *CA6* (rs2274327) were associated with picky eating behavior in children. There was no association between SNPs and BMI *z*-scores.

**Conclusion:** Genes related to chemosensory perception may play a role in children's picky eating behavior.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## 3.1 Introduction

Picky eating is a common problematic eating behavior among preschool-aged children (van der Horst et al., 2016). Although there is no operational definition of picky eating, the behavior is often characterized by strong food preferences and rejection of familiar or new foods (Taylor et al., 2015). Picky eating may influence the risk of both underweight and overweight (Dubois et al., 2007a; Finistrella et al., 2012), development of functional gastrointestinal distress (Tharner et al., 2015) and eating disorders later in childhood (Marchi & Cohen, 1990). In addition, picky eating in children is associated with emotional and behavioral problems, such as depression, anxiety, and somatic complaints (Machado et al., 2016; Segovia, 2015).

Picky eaters have specific food preferences and avoidances during mealtimes compared to non-picky eaters (Boquin et al., 2014b; Jacobi et al., 2008). These mealtime behaviors are associated with reduced dietary variety (Carruth et al., 1998; Jacobi et al., 2003), including lower intake of fruits (Galloway et al., 2005; Jacobi et al., 2008), vegetables (Cardona Cano et al., 2015; Galloway et al., 2005; Galloway et al., 2003; Jacobi et al., 2003; Tharner et al., 2014; van der Horst et al., 2016), whole grains (Cardona Cano et al., 2015; Tharner et al., 2014), dairy (Jacobi et al., 2008), and meat (Cardona Cano et al., 2015; Jacobi et al., 2008; Tharner et al., 2014; van der Horst et al., 2016). Twin studies in children indicate that picky eating behavior has considerable genetic contributions, with heritability estimates for food fussiness and food neophobia ranging from 0.46-0.78 (Cooke et al., 2007; Faith et al., 2013; Smith et al., 2017). Children have a biological predisposition for salty and sweet tastes, and an innate rejection for bitter and sour tastes (Birch, 1999; Drewnowski, 1997). Genetic variation in taste sensitivity may influence food acceptance and dietary intake in children (Keller et al., 2002; Mennella, 2005;

O'Brien et al., 2013; Turnbull & Matisoo-Smith, 2002), and could impact mealtime behaviors such as picky eating.

Although differences in taste perception are due to genetic variation (Dias et al., 2015; Hayes et al., 2015; Mennella, 2005; Padiglia et al., 2010), it is unclear whether these variations are related to picky eating behavior. The present study had 2 aims. First, we identified the influence of genetic variation on picky eating behavior in a sample of non-Hispanic white preschool-aged children. Specifically, we determined whether single nucleotide polymorphisms (SNPs) in genes related to chemosensory perception would predict children's limited dietary variety, food refusals, and struggles for control during mealtimes. Second, we tested the relationship between genetic variation and child BMI *z*-scores (BMIZ), which is considered an optimal measure for assessing adiposity at a single occasion (Cole et al., 2005). We hypothesized that variations in genes for chemosensory perception would be associated with picky eating behavior.

## 3.2 Methods

### Data and Participants

Data were drawn from a larger study, the Synergistic Theory and Research on Obesity and Nutrition Group (STRONG) Kids program, a prospective interdisciplinary research panel study aimed at identifying the multiple factors related to childhood obesity among preschool children (Harrison et al., 2011). Parents of children ($n$ = 153) aged 2-5 years were recruited from licensed childcare centers throughout east-central Illinois. Parents completed a questionnaire providing demographic data (age, gender, race/ethnicity, marital status, and education level) and information regarding their child's breastfeeding history and picky eating behavior. Surveys

were completed online or on paper if the participant did not have internet access. All parents of the children provided written informed consent. Child height, weight, and saliva samples were obtained by trained researchers either at the childcare center or in the family home. Analysis was restricted to non-Hispanic white children to control for effects of genetic ancestry. Children were excluded if they had a BMI <5th percentile to account for potential failure to thrive and underlying disease or illness. Less than 5% of the original sample ($n$ = 4) was excluded based on BMI percentile. This study was approved by the Institutional Review Board at the University of Illinois at Urbana-Champaign and met all requirements for ethical conduct for research with human subjects.

**Measures**

***Picky Eating Behavior***

Child picky eating behavior was assessed using the Oregon Research Institute Child Eating Behavior Inventory (ORI-CEBI), a validated tool that addresses problematic eating and feeding behaviors (Lewinsohn et al., 2005). The ORI-CEBI is comprised of 3 subscales that assess picky eating behavior, namely *limited variety*, *food refusals*, and *struggles for control*. The *limited variety* subscale examined parents' perceptions of their child's pickiness and intake of a limited variety of foods (4 items; Cronbach's $\alpha = 0.82$). The *food refusals* subscale examined parents' perception of their child's refusal to eat specific foods (12 items; Cronbach's $\alpha = 0.83$). The *struggles for control* subscale examined parents' perception of frequent struggles with the child over food (8 items; Cronbach's $\alpha = 0.88$). Parents were asked to rate the frequency of each picky eating behavior using a 5-point Likert-scale (1 = never, 3 = sometimes, 5 = always) with a not applicable option. A total score for each subscale was calculated by taking the mean of

item scores; high scores on the subscales represented higher levels of picky eating behavior. Picky eating was defined by the upper quartile of the data for each of the 3 subscales.

***Anthropometric Measures***

Children's height (cm) and weight (kg) were measured by trained personnel using a portable stadiometer (SECA, Model 242, Hanover, MD, USA) and an electronic remote display scale (Health-O-meter, Model 349KLX, Jarden Consumer Solutions, Boca Raton, FL, USA). The precision level for measurements was set to 0.1 cm for height and 0.1 kg for weight. To ensure accuracy, the measurements were taken 3 times, and the average of the 3 measurements was recorded. Child BMI was calculated as weight (kg)/height$^2$ ($m^2$) using the standard Statistical Analysis Software (SAS) Program from the Centers for Disease Control and Prevention (CDC), and converted into age and sex-specific body mass index (BMI) *z*-scores based on the 2000 CDC Growth Charts for the United States (CDC, 2000).

***DNA Extraction and Genotyping***

Child saliva samples were collected using the Saliva Collection Kit for Young Children Genomic DNA (DNA Genotek Inc., Kanata, Ontario, Canada) by trained personnel. Genomic DNA was extracted from saliva samples using protocol for purification of DNA from 4.0 mL Oragene DNA/saliva. Nine SNPs from 6 candidate genes that had been previously implicated in chemosensory perception were selected for analysis: rs2234233 (*TAS2R1)*; rs10772420 (*TAS2R19)*; rs713598, rs1726866, and rs10246939 (*TAS2R38)*; rs2274327 and rs2274333 (*CA6)*; rs34160967 (*TAS1R1)*; and rs35874116 (*TAS1R2)* (Calò et al., 2011; Dias et al., 2015; Hayes et al., 2013; Pirastu et al., 2012). SNPs were genotyped using Fluidigm® Genotyping Analysis version 4.1.2 (San Francisco, CA, USA). The minor allele frequencies (MAF) of all SNPs were within 5% of those reported in Caucasian populations (as reported for CEU HapMap population,

http://hapmap.ncbi.nlm.nih.gov/). Genotype frequencies for each SNP were evaluated for consistency with Hardy-Weinberg equilibrium (HWE) using a chi-square test. Two SNPs (rs1726866 and rs34160967) were in extreme HWE deviation ($p < 0.05$), and therefore, not included in the study. Linkage disequilibrium (LD) analyses was performed on *TAS2R38* and *CA6* (**Table 3.1**). The 3 *TAS2R38* variants associated with chemosensory perception were in strong LD ($R^2 = 0.57\text{-}0.89$, $D^2 = 0.89\text{-}1.0$). Thus, similar to previous studies, results are presented for rs713598 only (Bouthoorn et al., 2014; Mennella, 2005). The *CA6* variants were not in LD ($R^2 = 0.07$, $D^2 = 0.52$); thus, results are presented for both rs2273427 and rs2274333.

**Statistical Analysis**

For picky eating subscales, responses of "not applicable" were counted as missing data and not included in the calculation of mean item scores for picky eating behaviors. Picky eating behavior data had skewed distributions and were unaffected by transformation. Thus, the data were dichotomized based on the upper 25% of the data, and picky eating was defined by the upper quartile (limited variety score ≥3.250, food refusal score ≥2.167, and struggles for control score ≥2.625). BMI percentiles were categorized according to the CDC cut-off points: obese (BMI ≥95th percentile), overweight (BMI ≥85th percentile but <95th percentile), normal weight (BMI ≥5th percentile but <85th percentile), and underweight (BMI <5th percentile). Participants were classified into 2 groups of either overweight (by combining obese and overweight categories) or normal weight.

The $\chi^2$ test was used to evaluate the associations between picky eating and child sex, weight category, breastfed status, marital status, and parent education. Independent-sample t-test was used to evaluate differences in picky eating behaviors by child age, child BMI, and maternal age. Generalized linear models were used to examine the association of each SNP as the

predictor for BMIZ, and logistic regression was used for picky eating behaviors (limited variety, food refusals, and struggles for control). Each SNP was tested for allelic associations with picky eating behaviors and BMIZ under additive, dominant, and recessive models. BMI and picky eating behaviors were calculated separately as the outcome after adjusting for child age, sex, parent education level, and breastfed status. All data were analyzed using SAS version 9.4 (SAS Institute, Cary, NC, USA). Statistical significance was determined at $p < 0.05$. Sample characteristics are expressed as means ± standard deviations for continuous data, and frequencies are expressed as percentages for categorical data.

## 3.3 Results

### Population Characteristics and Prevalence of Picky Eating

Descriptive characteristics of the study sample are shown in **Table 3.2**. The children had a mean age of 37.5 ± 7.5 months, with about half being female (49.0%). The majority of children were normal weight with a mean BMIZ of 0.47 ± 0.8. Based on BMI percentiles, 24% of children were overweight or obese (i.e., BMI-for-age ≥85%). Almost a quarter of children were never breastfed. Most parents were female (92%), college graduates (63%) and married (82%), with a mean age of 32.9 ± 5.6 years. Picky eating behavior was prevalent in the study sample. Based on parental perception, 31.5% of children had limited dietary variety, 21% often refused food, and 25% had struggles for control during mealtimes. Initial analyses of potential covariates indicated no differences in picky eating behavior based on child age, sex, breastfed status, maternal age, or parent education level. When the children were classified as picky or non-picky eater by the survey question on limited dietary variety, there was a significant difference in BMIZ between picky eaters and non-picky eaters ($t = 2.14$, $p = 0.03$). Children with limited

dietary variety had significantly lower mean BMIZ (0.2770) compared to non-picky eaters (0.5862).

**Association between Genotype and Picky Eating Behaviors**

A total of 6 SNPs from 5 candidate genes were subsequently analyzed and there was no significant deviation from HWE (**Table 3.3**). The significant associations between genotype and picky eating are summarized in **Table 3.4**. There was a significant difference in limited variety according to genotype for the *TAS2R38*-rs713598 SNP ($p$ = 0.0345, recessive model). Children with at least one copy of the minor allele (C) of rs713598 were more likely to have limited dietary variety (OR = 2.31; 95% CI: 1.06-5.01) compared to children homozygous for the major allele (G) as shown in **Table 3.5**. There was a significant difference in struggles for control according to genotype for the *CA6*-rs2274327 SNP ($p$ = 0.500 and $p$ = 0.0290, for additive and dominant models, respectively). Children homozygous for the major allele (C) were more likely to have struggles for control during mealtimes (OR: 3.18; 95% CI: 1.13-8.98) compared to children with at least one copy of the minor allele (T). No other associations between genotype and picky eating were found when including child age, sex, breastfed status, and parent education level as covariates. **Tables 3.6-3.8** contain the associations between genotype and picky eating for all investigated SNPs.

**Association between Genotype and BMIZ**

The associations between genotype and BMIZ are shown in **Table 3.9**. There were no significant associations between genotype and BMIZ when including child age, sex, breastfed status, and parent education level as covariates.

### 3.4 Discussion

Of the 5 chemosensory genes evaluated, an association was observed between picky eating behavior and genetic variants in *TAS2R38* (taste receptor 2 member 38) and *CA6* (carbonic anhydrase VI, or gustin). The *TAS2R38*-rs713598 SNP was associated with limited dietary variety, and the *CA6*-rs2274327 SNP was associated with struggles for control during mealtimes. These findings support the hypothesis that variations in genes related to chemosensory perception are associated with picky eating behavior in preschool-aged children. Both *TAS2R38* and *CA6* have been proposed as genes related to bitter taste perception (Calò et al., 2011; Mennella, 2005). Previous research suggest that genetic sensitivity to bitter taste may influence dietary variety and food acceptance in children (Bell & Tepper, 2006; Keller et al., 2002). In the present study, children with limited dietary variety had a lower mean BMIZ compared to non-picky eaters, consistent with previous reports that picky eaters have lower body weight compared to non-picky eaters (Galloway et al., 2005; Rodenburg et al., 2012). Evidence suggests a relationship between inherited bitter sensitivity and weight status in preschoolers (Keller & Tepper, 2004; Lumeng et al., 2008). However, we found no association between variations in chemosensory genes and BMIZ.

One of the most widely studied genetically-inherited traits is the ability to taste bitter thiourea compounds, including 6-*n*-propylthiouracil (PROP) and phenylthiocarbamide (PTC) (Bufe et al., 2005). Although PROP and PTC are not present in foods, other thiourea-containing compounds (i.e., goitrins and sinigrins) are responsible for the bitter taste of cruciferous vegetables, such as broccoli, cabbage, and Brussels sprouts (Keller & Adise, 2016). Sensitivity to PROP and PTC is related to 3 functional variants in the *TAS2R38* bitter taste receptor gene that encode for amino acid substitutions: rs713598 (G145C, Ala49Pro), rs1726866 (T785C,

Val262Ala), and rs10246939 (A886G, Ile296Val) (Kim, 2003). The 3 SNPs contribute to 2 common haplotypes related to detection thresholds for bitter taste. The dominant taster haplotype (PAV, Pro-Ala-Val) confers bitter sensitivity, whereas the recessive non-taster haplotype (AVI, Ala-Val-Ile) is less functional (Bufe et al., 2005; Kim, 2003). The *TAS2R38*-rs713598 C allele is the bitter-sensitive allele, whereas the G allele is the bitter-insensitive allele (Kim, 2003). Children with the bitter-sensitive allele are reported to consume more added sugar, and prefer cereals and beverages with significantly higher sugar content compared to children with the bitter-insensitive allele (Mennella, 2005). Interestingly, picky eaters consume more confectionary items compared to non-picky eaters (Cardona Cano et al., 2015; Tharner et al., 2014). In the present study, having at least one copy of the rs713598 C allele was associated with increased risk of limited dietary variety, suggesting that these bitter-sensitive children, potentially supertasters (Bartoshuk et al., 1994; Feeney et al., 2011), are more likely to be picky eaters.

Variations in chemosensory genes can also influence taste perception by altering salivary proteins and fungiform papillae density on the tongue (Feeney et al., 2011; Hayes et al., 2013). Gustin, a salivary protein encoded by the *CA6* gene, influences taste function by binding to zinc at the active site (Shatzman & Henkin, 1981). Padiglia et al. (2010) found that polymorphisms in the *CA6* gene influence the binding of gustin and zinc, thus impacting taste function. In addition, bitter taste sensitivity is inversely associated to fungiform papillae density (Bartoshuk et al., 1994; Essick et al., 2003), and gustin is a reported trophic factor involved in taste bud growth and development (Barbarossa et al., 2015; Calò et al., 2011; Melis et al., 2013). We found that homozygosity of the *CA6*-rs227432 C allele was associated with increased risk of struggles with control during mealtimes, suggesting that gustin polymorphisms may play a role in children's

picky eating behavior. Previous studies have shown an association between the *CA6*-rs227432 T allele and lower salivary buffer capacity (Aidar et al., 2013; Peres et al., 2010).

Child eating behavior is influenced by sensory sensitivity to various stimuli (Farrow & Coulthard, 2012), and chemosensory factors other than taste may play a role in picky eating. For some picky eaters, the type and presentation of food are the strongest drivers of mealtime behavior and food consumption (Boquin et al., 2014a). Children's food acceptance and dietary intake are influenced by sensory properties of food, such as odor (Monnery-Patris et al., 2015), color (Addessi et al., 2005), and texture (Coulthard et al., 2009; Coulthard & Thakker, 2015; Werthmann et al., 2015). Children who are picky eaters may be more sensitive to olfactory cues (Monnery-Patris et al., 2015) and tactile stimuli (Nederkoorn et al., 2015), which could influence their intake and liking of foods. Future studies are needed to understand how children's food preferences and eating behaviors are shaped by the appearance, texture, and smell of food.

This study has limitations that should be considered when interpreting the results. First, the small sample size may lack sufficient statistical power to determine all the associations between genetic variations and picky eating behaviors investigated in the study. Larger studies may aid to verify the present results and detect other associations. Second, this study only focused on non-Hispanic white children from predominantly well-educated families. Thus, our results may not be generalizable to other racial/ethnic groups or those of lower socioeconomic status. Finally, we did not assess the PROP taster phenotype, which could be related to genotype and picky eating.

## 3.5 Conclusion

In conclusion, picky eating behavior is a common mealtime problem among young children that is influenced by a child's genetic background. The current study suggests that genetic variation in *TAS2R38* and *CA6* influence picky eating in preschoolers. Additional work is needed to confirm whether variations in chemosensory genes are associated with differences in children's long-term eating behavior and weight trajectory.

### 3.6 Tables

**Table 3.1.** Linkage Disequilibrium (D' and $R^2$) by Composite Haplotype Method

| **Gene** | **SNP** | **rs10246939** | **rs1726866** | **rs713598** |
|---|---|---|---|---|
| ***TAS2R38*** | **rs10246939** | **-** | D' = 1 | D' = 1 |
| | **rs1726866** | $R^2$ = 0.5671 | **-** | D' = 0.8931 |
| | **rs713598** | $R^2$ = 0.8572 | $R^2$ = 0.4817 | **-** |
| | | | | |
| | | | | |
| **Gene** | **SNP** | **rs2274327** | **rs2274333** | |
| ***CA6*** | **rs2274327** | **-** | D' = 0.5224 | |
| | **rs2274333** | $R^2$ = 0.07446 | **-** | |

**Table 3.2.** Characteristics of study sample (*n* = 153)

| | |
|---|---|
| **Child's characteristics** | |
| Age (months) | 37.5 ± 7.5 |
| Female | 75 (49.0) |
| BMIZ | 0.47 ± 0.8 |
| BMI percentile[a] | |
| Normal weight | 116 (75.8) |
| Overweight | 37 (24.2) |
| Breastfed | 116 (75.8) |
| Picky eating behavior[b] | |
| Limited variety | 47 (31.5) |
| Food refusal | 32 (21.2) |
| Struggles for control | 38 (25.3) |
| **Parent's characteristics** | |
| Age (years) | 32.9 ± 5.6 |
| Female | 140 (91.5) |
| Married | 126 (82.4) |
| Education | |
| Grade school/high school | 14 (9.1) |
| College/technical school | 39 (25.5) |
| College graduate/postgraduate work | 97 (63.4) |
| Unknown/missing | 3 (2.0) |

Data are presented as *n* (%) or mean ± SD, BMIZ, BMI z-score. [a] Based on CDC cut-off: overweight, BMI ≥ 85th percentile; normal weight (BMI ≥ 5th percentile but <85th percentile). [b] Upper quartile (Limited Variety Score ≥ 3.250, Food Refusal Score ≥ 2.167, Struggles for Control Score ≥ 2.625).

**Table 3.3.** Characteristics of SNPs

| Gene | SNP | Chromosome: position, kb | Minor allele frequency reported[a] | SK Cohort | HWE *p* value |
|---|---|---|---|---|---|
| *TAS2R1* | rs2234233 | 5: 9629417 | T 0.18 | 0.16 | 0.45 |
| *TAS2R19* | rs10772420 | 12: 11021677 | A 0.47 | 0.44 | 0.44 |
| *TAS2R38* | rs713598 | 7: 141973545 | C 0.43 | 0.44 | 0.29 |
| *CA6* | rs2274327 | 1: 8949347 | T 0.36 | 0.39 | 0.60 |
| | rs2274333 | 1: 8957145 | G 0.31 | 0.27 | 0.55 |
| *TAS1R2* | rs35874116 | 1: 18854899 | C 0.31 | 0.33 | 0.15 |

HWE = Hardy-Weinberg equilibrium; SK= STRONG Kids. [a] Caucasian population (HAPMAP).

**Table 3.4.** Significant associations between genotype and picky eating behavior

| | | Picky eating behavior | | |
|---|---|---|---|---|
| **SNP** | **Model**[a] | **limited variety** | **food refusals** | **struggles for control** |
| rs713598 | Additive | 0.0766 | 0.3764 | 0.1002 |
| | Dominant | 0.1189 | 0.9436 | 0.3510 |
| | Recessive | **0.0345** | 0.1873 | 0.1417 |
| rs2274327 | Additive | 0.2825 | 0.2732 | **0.0500** |
| | Dominant | 0.1134 | 0.1334 | **0.0290** |
| | Recessive | 0.7262 | 0.9159 | 0.0831 |

Bolded *p* values are significant at $\alpha = 0.05$. [a] Adjusting for child age, gender, breastfed status, parent education level.

**Table 3.5.** Odds ratios for picky eating behavior according to genotype for rs713598 and rs2274327

| SNP | Genotype | Non-picky eaters, n (%) | Picky eaters, n (%)[a] | Odds ratio (95% CI)[b] |
|---|---|---|---|---|
| rs713598[c] | GG | 26 (17.5) | 7 (4.7) | 0.47 (0.18-1.22) |
| | CG/CC | 76 (51.0) | 40 (26.8) | **2.31 (1.06-5.01)** |
| rs2274327[d] | CC | 46 (30.7) | 9 (6.0) | **3.18 (1.13-8.98)** |
| | CT/TT | 66 (44.0) | 29 (19.3) | 0.47 (0.20-1.10) |

Bolded *p* values are significant at $\alpha = 0.05$.[a] Based on upper quartile. [b] Adjusting for child age, gender, breastfed status, parent education level. [c] Results listed are for limited variety. [d] Results listed are for struggles for control.

**Table 3.6.** Association between genotype and limited variety

| SNP | Genotype | LSMeans ± se | PE Association Model[a], p-value | | |
|---|---|---|---|---|---|
| | | | Additive | Dominant | Recessive |
| rs2234233 | CC | -0.993 ± 0.306 | 0.3812 | 0.2367 | 0.6937 |
| | CT | -1.315 ± 0.458 | | | |
| | TT | 0.188 ± 1.045 | | | |
| rs10772420 | AA | -0.988 ± 0.387 | 0.7824 | 0.4947 | 0.9535 |
| | AG | -0.922 ± 0.340 | | | |
| | GG | -1.294 ± 0.522 | | | |
| rs713598 | GG | -0.458 ± 0.373 | 0.0766 | 0.1189 | **0.0345** |
| | GC | -1.150 ± 0.368 | | | |
| | CC | -1.617 ± 0.491 | | | |
| rs2274327 | CC | -1.146 ± 0.384 | 0.2825 | 0.1134 | 0.7262 |
| | CT | -1.208 ± 0.361 | | | |
| | TT | -0.351 ± 0.489 | | | |
| rs2274333 | AA | -1.039 ± 0.323 | 0.9702 | 0.9630 | 0.8075 |
| | AG | -0.944 ± 0.380 | | | |
| | GG | -0.968 ± 0.665 | | | |
| rs35874116 | TT | -0.893 ± 0.340 | 0.2882 | 0.1147 | 0.5057 |
| | TC | -0.899 ± 0.360 | | | |
| | CC | -1.949 ± 0.682 | | | |

Bolded *p* values are significant at $\alpha = 0.05$.[a] Adjusting for child age, gender, breastfed status, parent education level.

**Table 3.7.** Selected SNPs and associations with food refusals

| SNP | Genotype | LSMeans ± se | PE Association Model[a], p-value | | |
|---|---|---|---|---|---|
| | | | Additive | Dominant | Recessive |
| rs2234233 | CC | -1.537 ± 0.338 | 0.6099 | 0.7178 | 0.3233 |
| | CT | -1.120 ± 0.454 | | | |
| | TT | -0.984 ± 1.195 | | | |
| rs10772420 | AA | -1.338 ± 0.418 | 0.9607 | 0.8748 | 0.7855 |
| | AG | -1.446 ± 0.379 | | | |
| | GG | -1.493 ± 0.563 | | | |
| rs713598 | GG | -1.072 ± 0.401 | 0.3764 | 0.9436 | 0.1873 |
| | GC | -1.721 ± 0.416 | | | |
| | CC | -1.445 ± 0.490 | | | |
| rs2274327 | CC | -1.467 ± 0.427 | 0.2732 | 0.1334 | 0.9159 |
| | CT | -1.743 ± 0.410 | | | |
| | TT | -0.811 ± 0.495 | | | |
| rs2274333 | AA | -1.414 ± 0.352 | 0.4517 | 0.2391 | 0.9821 |
| | AG | -1.224 ± 0.406 | | | |
| | GG | -2.587 ± 0.074 | | | |
| rs35874116 | TT | -1.327 ± 0.374 | 0.4653 | 0.2162 | 0.6416 |
| | TC | -1.315 ± 0.391 | | | |
| | CC | -2.286 ± 0.790 | | | |

Bolded *p* values are significant at $\alpha = 0.05$.[a] Adjusting for child age, gender, breastfed status, parent education level.

**Table 3.8.** Selected SNPs and associations with struggles for control

| SNP | Genotype | LSMeans ± se | PE Association Model[a], p-value | | |
|---|---|---|---|---|---|
| | | | Additive | Dominant | Recessive |
| rs2234233 | CC | -1.225 ± 0.323 | 0.4037 | 0.1785 | 0.7666 |
| | CT | -1.256 ± 0.461 | | | |
| | TT | 0.213 ± 1.062 | | | |
| rs10772420 | AA | -1.123 ± 0.414 | 0.9380 | 0.8186 | 0.8476 |
| | AG | -1.246 ± 0.362 | | | |
| | GG | -1.083 ± 0.509 | | | |
| rs713598 | GG | -0.814 ± 0.396 | 0.1002 | 0.3510 | 0.1417 |
| | GC | -1.732 ± 0.414 | | | |
| | CC | -0.858 ± 0.443 | | | |
| rs2274327 | CC | -1.790 ± 0.454 | **0.0500** | **0.0290** | 0.0831 |
| | CT | -1.251 ± 0.379 | | | |
| | TT | -0.305 ± 0.485 | | | |
| rs2274333 | AA | -1.450 ± 0.355 | 0.2981 | 0.6006 | 0.1199 |
| | AG | -0.832 ± 0.387 | | | |
| | GG | -0.807 ± 0.664 | | | |
| rs35874116 | TT | -1.274 ± 0.374 | 0.9025 | 0.9361 | 0.6587 |
| | TC | -1.085 ± 0.373 | | | |
| | CC | -1.146 ± 0.574 | | | |

Bolded *p* values are significant at $\alpha = 0.05$.[a] Adjusting for child age, gender, breastfed status, parent education level.

**Table 3.9.** Selected SNPs and associations with BMIZ

| SNP | Genotype | LSMeans ± se | BMIZ Association Model[a], p-value | | |
|---|---|---|---|---|---|
| | | | Additive | Dominant | Recessive |
| rs2234233 | CC | 0.549 ± 0.101 | 0.2079 | 0.0961 | 0.8977 |
| | CT | 0.645 ± 0.155 | | | |
| | TT | -0.125 ± 0.414 | | | |
| rs10772420 | AA | 0.487 ± 0.137 | 0.4923 | 0.4395 | 0.5325 |
| | AG | 0.628 ± 0.118 | | | |
| | GG | 0.435 ± 0.176 | | | |
| rs713598 | GG | 0.517 ± 0.137 | 0.3224 | 0.2273 | 0.6997 |
| | GC | 0.657 ± 0.123 | | | |
| | CC | 0.399 ± 0.155 | | | |
| rs2274327 | CC | 0.670 ± 0.132 | 0.4038 | 0.7062 | 0.1774 |
| | CT | 0.481 ± 0.119 | | | |
| | TT | 0.487 ± 0.186 | | | |
| rs2274333 | AA | 0.536 ± 0.111 | 0.5805 | 0.3615 | 0.8508 |
| | AG | 0.610 ± 0.132 | | | |
| | GG | 0.353 ± 0.237 | | | |
| rs35874116 | TT | 0.541 ± 0.122 | 0.3284 | 0.1510 | 0.8466 |
| | TC | 0.479 ± 0.123 | | | |
| | CC | 0.792 ± 0.193 | | | |

Bolded *p* values are significant at $\alpha = 0.05$.[a] Adjusting for child age, gender, breastfed status, parent education level.

# CHAPTER 4:
# Home Feeding Environment and Picky Eating Behavior in Preschool-Aged Children: A Prospective Analysis[4]


## Abstract

**Objective:** Picky eating is common in children and can persist into adolescence and adulthood. Identifying predictors of picky eating could inform the development of anticipatory feeding guidance for parents and caregivers of young children. This study identified factors related to the home feeding environment in the prediction of picky eating behavior in a cohort of preschool-aged children.

**Methods:** Parents of preschool-aged children (n = 497) completed questionnaires including measures of the home feeding environment (i.e., television during mealtime, family mealtime routines, and feeding practices) and child picky eating behavior. The questionnaire was repeated 1 year later, in which 326 parent-child dyads participated. Logistic regression was used to determine the cross-sectional and prospective associations between home feeding environment measures and child picky eating behavior outcomes.

**Results:** Child control over feeding and watching television during mealtime was associated with higher odds of picky eating behavior in both cross-sectional and prospective analyses. Family mealtime routine was associated with lower odds of picky eating behavior 1 year later.

**Conclusion:** The home feeding environment plays a role in the development of young children's picky eating behavior. Avoiding the television and maintaining parent control of food choices during mealtimes could lead to improvements in children's picky eating behavior.


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[4] Cole, N. C., Musaad, S. M., Lee, S. Y., & Donovan, S. M. (2017). Home feeding environment and picky eating behavior in preschool-aged children: a prospective analysis. *Under Review*.

## 4.1 Introduction

Picky eating behavior in children is characterized by the avoidance of food and inadequate dietary variety (Taylor et al., 2015), often causing concern and stress for parents during mealtimes (Goh & Jacob, 2012; Trofholz et al., 2017). These mealtimes behaviors are prevalent among young children – an estimated 21 to 50% of children in the United States between the ages of two and five years are reported by their parents as being picky eaters (Carruth et al., 2004a; Jacobi et al., 2003; Mascola et al., 2010). Picky eating in children has been associated with nutrient deficiencies (Taylor et al., 2016; Xue et al., 2015) and behavioral problems (Cardona Cano et al., 2016; Jacobi et al., 2008), and can persist into adolescence and adulthood (Mascola et al., 2010; Van Tine et al., 2017). Researchers have identified several factors related to the home feeding environment that may contribute to picky eating behavior in children. However, few studies have examined the longitudinal impact of the home feeding environment on picky eating behavior in children under five years of age.

It is widely accepted that children have more positive eating behaviors when parents are responsible for what, when, and where their child eats, while children are responsible for deciding whether to eat and how much (Danaher & Fredericks, 2012; Dietz & Stern, 1999; Satter, 1990). A responsive feeding environment supports the child's hunger and satiety cues and ensures a pleasant and structured feeding environment with few distractions (Black & Aboud, 2011). Supportive and structured mealtimes tend to promote healthy eating behavior and weight in children (Satter, 1995; Satter, 1986), and are negatively associated with picky eating behavior (Finnane et al., 2017; Powell et al., 2017). A nonresponsive feeding environment lacks reciprocity between the parent and child, does not establish eating in the same place, and can override the child's hunger and satiety cues (Black & Aboud, 2011; DiSantis et al., 2011).

Nonresponsive feeding practices, including parental control over eating (e.g., pressure to eat and overt restriction), are associated with picky eating in children (Finnane et al., 2017; Galloway et al., 2003; Jansen et al., 2014).

Previous research relating picky eating and the home feeding environment have primarily been cross-sectional studies. A small number of studies have examined longitudinal effects of parent feeding practices on picky eating behavior, and have found that picky eating is related to pressure to eat (Antoniou et al., 2016; Berger et al., 2016; Galloway et al., 2005). However, these studies were conducted in older school-aged children. With regards to supportive and structured mealtimes, cross-sectional studies in young children have shown that picky eating is negatively associated with family meal settings (Jansen et al., 2014) and mealtimes with no distractions, such as the television (Powell et al., 2017). Research is needed to examine if picky eating is related to the home environment in younger samples. Identifying predictors of picky eating in younger children could inform the development of anticipatory feeding guidance for parents and caregivers.

The objective of the present study was to examine the association between factors of the home feeding environment with picky eating behavior outcomes in a sample of preschool-aged children (two to five years) observed at two-waves (baseline and one-year follow-up). We hypothesized that a nonresponsive home feeding environment (i.e., television viewing during mealtimes, lesser adherence to family mealtime routines, and nonresponsive feeding practices) at baseline would be associated with higher picky eating behavior in children at baseline and one-year follow-up.

## 4.2 Methods

### Data and Participants

Data were drawn from the first two waves of the Synergistic Theory and Research on Obesity and Nutrition Group (STRONG) Kids program, a three-wave prospective panel study conducted annually aimed at identifying the biological and environmental factors that predict eating habits and obesity risk in preschool-aged children (Harrison et al., 2011; Harrison & Liechty, 2012). Participants were recruited from 30 licensed childcare centers in east-central Illinois. Most primary caregivers were the child's biological parent (93.4%), thus the term 'parent' is used hereafter. Parents completed a comprehensive panel survey (online or on paper) at baseline and at one-year follow-up and were compensated $50 for completing each survey. All parents provided written informed consent. This study was approved by the Institutional Review Board at the University of Illinois at Urbana-Champaign and met all requirements for ethical conduct for research with human subjects.

### Measures

#### *Child Picky Eating Behavior*

The outcome of picky eating behavior was assessed using two subscales from the Oregon Research Institute Child Eating Behavior Inventory (ORI-CEBI) (Lewinsohn et al., 2005). The *limited variety* subscale examined parental perception of their child's picky eating and intake of a limited variety of foods (five items; Cronbach's $\alpha = 0.76$). The *food refusals* subscale examined parental perception of their child's refusal to eat 12 food items (fish, soups, vegetables, fruit, meats, eggs, cheese, milk, yogurt, potatoes/rice/pasta, breads, and cereals). Cronbach's alpha for the current sample was high (0.89). Parents reported the frequency of their child's picky eating

using a 5-point Likert-scale (1 = never to 5 = always) with a not applicable option that was not included in scoring. Higher mean scores indicated more picky eating behavior.

***Home Feeding Environment***

Three characteristics of the home feeding environment were assessed as independent variables: television during mealtime, family mealtime routine, and feeding practices.

Television during mealtime was measured using three individual survey questions. To assess the presence of television during mealtime, parents were asked the questions "Is there a television in the kitchen?" and "Is a television in view where your family eats most meals?" using yes/no responses (0 = no, 1 = yes). To assess the frequency of television use during mealtime, parents were asked "How often is the TV on in the space where your family is eating meals?" using a 5-point Likert scale (1 = never to 5 = all the time). Scores were dichotomized by coding responses of 1 as 'no television use during mealtime' versus grouping responses of 2-5 as 'any television use during mealtime'.

Family mealtime routines were measured using an adapted scale (Jones et al., 2014) from the Family Ritual Questionnaire (Fiese & Kline, 1993), and was calculated by averaging five items asking parents about their family mealtimes using a 5-point Likert scale (1 = not true at all to 5 = very true): 1) "In our family, mealtime is planned in advance;" 2) "Our family regularly eats the main meal together;" 3) In our family, everyone is expected to be home for the main meal;" (4) "In our family at mealtime, everyone has a specific role or job to do;" and the reverse-coded item (5) "In our family, mealtime is flexible; people eat whenever they want". Cronbach's alpha for the current sample was good (0.75). Higher scores on the scale represented a higher sense of positive climate during family meals and mealtime ritualization.

Feeding practices were measured using the Comprehensive Feeding Practices Questionnaire (CFPQ), which is comprised of 49 items asking parents to indicate how often they use specific feeding practices (Musher-Eizenman & Holub, 2007). Each item was measured using a 5-point Likert-type scale (1 = never to 5 = always). Higher scores represented higher use of that feeding practice. The 12 subscales included were calculated by averaging responses for specific items: monitoring (keeping track of child's food consumption; four items), environment (making healthy foods available in the home; four items), child control (allowing child control of food choices, parent-child feeding interactions, and eating behaviors at the dinner table; five items), emotion regulation (using food to regulate the child's emotions; three items), balance and variety (promoting well-balanced food intake; four items), food reward (using food as a reward for good behavior; three items), involvement (encouraging child's involvement in meal planning and food preparation; three items), modeling (actively demonstrating healthy eating; four items), pressure (pressuring child to eat during meals; four items), restriction for health (restricting child's food intake for health; four items), restriction for weight (controlling child's food intake to decrease or maintain weight; eight items), and teaching about nutrition (using didactic techniques to encourage child's intake of healthy foods; three items). Based on responsive/child-centered and nonresponsive/parent-centered feeding directives as previously described (Black & Aboud, 2011; Hughes et al., 2006; Hughes et al., 2005), nonresponsive feeding practices in the CFPQ include child control, emotion regulation, food reward, pressure, restriction for health, and restriction for weight, whereas responsive feeding practices include monitoring, environment, balance and variety, involvement, modeling, and teaching about nutrition. Cronbach's alphas showed acceptable to good reliability in the current sample—between 0.66 and 0.89 for most subscales except for the child control subscale (alpha of 0.57). Although the child control

subscale had questionable reliability, it was retained because it assessed constructs of interest in this study and to provide preliminary information on the relation with picky eating.

### *Control Variables*

Age and sex-specific body mass index *z*-scores (BMIZ) were calculated from the child's height and weight measurements based on the 2000 Centers for Disease Control and Prevention Growth Charts for the United States (CDC, 2000). Trained research personnel measured the children's height (cm) and weight (kg) using a portable stadiometer (SECA, Model 242, Hanover, MD, USA) and an electronic remote display scale (Health-O-meter, Model 349KLX, Jarden Consumer Solutions, Boca Raton, FL, USA). Additional control variables included the child's age, sex, race/ethnicity, breastfeeding history, and parent education level. Baseline picky eating behavior was controlled for in prospective analyses.

## Statistical Analyses

All statistical analyses were completed using Statistical Analysis Software (SAS, version 9.4, 2012, SAS Institute Inc., Cary, NC). Statistical significance was set to a two-tailed $p < .05$. The analytic strategy was to identify cross-sectional and prospective associations among independent variables of the home feeding environment (television during mealtime, family mealtime routine, and feeding practices) and outcome variables of child picky eating (limited variety and food refusals). Both picky eating outcome variables had skewed distributions and were unaffected by transformation. To address this problem and to compare extremes in picky eating behavior, mean scores for the limited variety and food refusals subscales were dichotomized into picky and non-picky based on the upper quartile ($75^{th}$ percentile) of the data as done in a previous publication (Cole et al., 2017b).

Polychoric correlations were conducted to determine the associations among categorical variables (i.e., two picky eating outcome variables and three television variables). Spearman's Rank correlations were conducted to determine the association between continuous home feeding environment variables (i.e., family mealtime routine and feeding practices). Logistic regression analyses were conducted to determine the association between the home feeding environment and each picky eating outcome at baseline (cross-sectional analysis) and at one-year follow-up (prospective analysis). The first model tested the independent effects of each home feeding environment measure (television during mealtime, family mealtime routine, and feeding practices) while adjusting for the control variables (child age, sex, BMIZ, race/ethnicity, breastfeeding history, and parent education). For prospective analyses, relations included only the subsample that completed assessments at both time points (n = 326) and the model controlled for baseline picky eating behavior. The second model tested the effect of all home feeding environment measures at the same time while also adjusting for the control variables.

## 4.3 Results

### Sample Characteristics

**Table 4.1** contains the sample characteristics at baseline and one-year follow-up. There were no significant differences in parent sex, marital status, parent education, child sex, child race/ethnicity, or breastfeeding status between baseline and one-year follow-up. The baseline sample comprised of 497 parent-child dyads, of which 326 (66%) participated in the one-year follow-up. Comparison studies had a higher follow-up rate (78-85%) at one-year (Gregory et al., 2010; Rodgers et al., 2013). There was no difference between completers and non-completers for any of the constructs investigated.

**Table 4.2** presents the bivariate correlations between the home feeding environment variables at baseline including descriptive statistics (i.e., means and standard deviations for continuous variables, and n (%) for categorical variables). The most frequently used feeding practice was monitoring the child's food consumption and providing balance and variety during meals. Whereas, using food to regulate the child's emotions and restricting food intake for weight control were less used by parents. In general, responsive feeding practices were highly correlated.

**Cross-Sectional Associations between Home Feeding Environment and Picky Eating**

Watching television during meals was associated with higher odds of limited variety (OR: 3.24; 95% CI: 1.29-8.17) and food refusals (OR: 2.61; 95% CI: 1.11-6.13). Parents that allowed child control over feeding were more likely to perceive their child as being a picky eater in terms of limited variety (OR: 2.32; 95% CI: 1.29-4.20) and refusal to eat specific foods (OR: 3.09; 95% CI: 1.74-5.48). Restriction for health was associated with higher odds of limited variety (OR: 1.68; 95% CI: 1.10-2.58).

Several measures of the home feeding environment were associated with lower odds of food refusals when controlling for demographics, child BMIZ, and breastfeeding history, including family mealtime routine (OR: 0.66; 95% CI: 0.48-0.91), balance and variety (OR: 0.60; 95% CI: 0.40-0.88), and involvement in meal planning or food preparation (OR: 0.68; 95% CI: 0.52-0.90). Providing a healthy feeding environment was associated with lower odds of limited variety (OR: 0.51; 95% CI: 0.32-0.80) and food refusals (OR: 0.58; 95% CI: 0.38-0.88). Additionally, parents that used food to regulate their child's emotions were more likely to perceive their child as a picky eater for limited variety (OR: 1.62; 95% CI: 1.07-2.45) and refusal

to eat specific foods (OR: 1.94; 95% CI: 1.30-2.87). However, these findings were no longer significant when controlling for other home feeding environment measures.

**Prospective Associations between Home Feeding Environment and Picky Eating**

**Table 4.3** presents prospective analyses of the home feeding environment measures at baseline and the picky eating behavior outcomes at one-year follow-up. Children whose parents promoted balance and variety during meals were more likely to be perceived as having limited dietary variety. Child control over feeding predicted higher odds of food refusals one year later, whereas emotion regulation predicted lower odds of food refusals. Similar to findings from cross-sectional analyses, watching television during meals predicted parental perception of limited variety. When controlling for demographics, child BMIZ, and breastfeeding history, children who had the television in view during meals were more than twice as likely to be perceived as a picky eater for limited variety and food refusals. Family mealtime routine was associated with lower odds of limited variety. However, this was no longer significant when controlling for other home feeding environment measures.

## 4.4 Discussion

This study supports the hypothesis that a nonresponsive home feeding environment (i.e., an environment that overrides child's hunger and satiety cues, lacks reciprocity between parent and child, and does not establish eating in the same place) predicts higher picky eating behavior in preschool-aged children. Specifically, child control over feeding (i.e., allowing child control of food choices, parent-child feeding interactions, and eating behaviors at the dinner table) and watching television during mealtime predicted higher odds of picky eating one year later.

It is widely known that parents' use of controlling feeding practices is related to children's picky eating (Mehta et al., 2014; Moroshko & Brennan, 2013; van der Horst, 2012; Webber et al., 2010). However, less is known about children's control over their food choices and feeding interactions. While child control might seem like an autonomy-promoting feeding practice, results from the present study suggest that when children control the mealtime, they are more likely to become picky eaters. Children can be involved in food preparation, but the decision of *what* to eat and *when* to eat should be set by the parents. This allows children to have autonomy during mealtime without compromising structure and consistency.

Previous research indicates that television viewing is associated with differences in food intake (Coon et al., 2001; Wenhold & Harrison, 2017). The television is considered a mealtime distraction and past studies have shown a positive association between picky eating in children with television viewing (Jacobi et al., 2003; Powell et al., 2017). In line with these findings, the present study indicates that watching television during meals is associated with more than two times higher odds of child limited variety in both cross-sectional and prospective analyses, even when controlling for other factors related to the home feeding environment. Additionally, having the television in view during meals at baseline was independently associated with higher odds of picky eating at one-year follow-up. This relation was not observed in the cross-sectional analysis. Collectively, these findings suggest that the mere presence of a television around mealtimes distracts children and negatively influences their eating behavior, and the effect may be more pronounced as children get older. One study in Singapore found that some parents may actually allow television viewing at mealtimes as a coping strategy for children's picky eating behavior (Goh & Jacob, 2012). Parents should be encouraged to maintain a distraction-free mealtime environment to support the development of healthy eating behaviors.

Family mealtime routine was associated with lower odds of picky eating behavior outcomes at one-year follow-up. This finding aligns with a recent study, in which mealtime ritualization was positively associated with healthy child behaviors (Musaad et al., 2017). It is known that involvement of parents in family meals, in terms of parent presence and frequency, is positively associated with healthy dietary intake in children, particularly for fruits, vegetables, whole-grains, and calcium-rich foods (Larson et al., 2006; Neumark-Sztainer et al., 2003; Videon & Manning, 2003). A recent longitudinal study found that higher levels of parent structuring/scaffolding (i.e., the parent's capacity to support the child's learning) reduced the risk of children's picky eating, suggesting that structuring the child's feeding environment may facilitate learning and exploration of a wide variety of foods (Steinsbekk et al., 2017). Findings from a recent qualitative study indicate that picky eating impacts the family meal by increasing the amount of time parents spend on meal preparation (Trofholz et al., 2017). In the present study, involving the child in meal planning and food preparation was associated with lower picky eating behavior, albeit its effect was only seen in the cross-sectional analysis. Involving the entire family in kitchen activities may benefit eating habits and the mealtime environment of both the child and the parent.

There were two predictors of picky eating in younger children from this study that were contrary to the hypothesis. First, parents that promoted balance and variety during meals (a responsive feeding practice) were more likely to perceive their child as a picky eater one year later. One potential reason for this finding is that the more dietary variety parents offer to their children, the more food options there are for the child to decide on, and potentially dislike or refuse. Parents may perceive their child as being pickier when they promote well-balanced food intake simply due to the increased difficulty in getting their child to eat from more numerous

choices. Another possibility is that parents may provide more diverse options during meals in response to the child's picky eating as an attempt to offer more food choices. The second finding was that parental use of food to regulate the child's emotional state (a nonresponsive feeding practice) demonstrated a protective effect against the development of food refusals. Using food for emotion regulation purposes (e.g., when the child is upset or fussy) is positively associated with child intake of candy/sweets (Blissett et al., 2010; Kiefner-Burmeister et al., 2014), and this is a food item that was not assessed by the ORI-CEBI food refusals subscale. Previous studies in children have found no association between emotion regulation and food fussiness or food neophobia (Powell et al., 2011; Tan & Holub, 2012).

Strengths of this study include the breadth of home feeding environment measures that were examined and a relatively diverse sample in terms of racial/ethnic groups. However, this study has some limitations. Measures of the home feeding environment and picky eating behavior relied on parent self-report and may be subject to social desirability bias. Also, other measures of screen time (e.g., computer/laptop, tablet device, smartphone) were not assessed. Responses were from only one parent (primarily the mother) and it would be beneficial to gather information from both parents to compare differences in feeding characteristics and perception of child picky eating behavior. Although this study utilized a longitudinal design with measures repeated at two time points one year apart, associations could be due to other factors not included in the model or that can only be detected at a farther time point. Finally, this is a relatively small sample of parents that may not be representative of other income or education levels. Further examination of these findings over a wider time period and in larger samples is warranted.

## 4.5 Conclusion

The home feeding environment is related to the development of picky eating behavior in preschool-aged children, particularly the presence and use of television during mealtimes and the allowance of children to control their food choices. To support the development of child autonomy, parents should provide a structured and supportive feeding environment for their children by choosing the food that is served at meals and turning off distractions during mealtime such as the television. Additional research is needed to identify factors in the home feeding environment that promote healthy food preferences and dietary intake in young children and prevent the development of problematic eating behavior.

## 4.6 Tables

**Table 4.1.** Sample characteristics

| | **Baseline (n = 497)** Number (%) or mean ± SD | **One-year follow-up (n = 326)** Number (%) or mean ± SD |
|---|---|---|
| **Parent characteristics** | | |
| Age, years | 32.4 ± 6.7 | 34.2 ± 6.5 |
| Female | 444 (89.3) | 299 (91.7) |
| Marital status | | |
| Single | 117 (23.5) | 61 (18.7) |
| Married, civil union, co-habitating | 343 (69.0) | 245 (75.2) |
| Separated, divorced, widowed | 30 (6.0) | 17 (5.2) |
| Unknown/missing | 7 (1.4) | 3 (.9) |
| Education | | |
| College graduate or higher | 268 (53.9) | 207 (63.5) |
| College/technical school | 159 (32.0) | 91 (27.9) |
| Grade school/high school | 68 (13.7) | 27 (8.3) |
| Unknown/missing | 2 (.4) | 1 (.3) |
| **Child characteristics** | | |
| Age, months | 39.0 ± 8.2 | 52.4 ± 8.4 |
| Female | 254 (51.1) | 158 (48.5) |
| BMIZ | .34 ± .97 | .41 ± 1.12 |
| Race/ethnicity | | |
| Non-Hispanic White | 278 (55.9) | 199 (61.1) |
| Non-Hispanic Black | 131 (26.4) | 75 (23.0) |
| Hispanic | 34 (6.8) | 18 (5.5) |
| Other | 54 (10.9) | 34 (10.4) |
| Breastfeeding history | | |
| Never breastfed | 137 (27.6) | 77 (23.6) |
| Breastfed < 6 months | 179 (36.0) | 119 (36.5) |
| Breastfed ≥ 6 months | 181 (36.4) | 130 (39.9) |
| Picky eating behavior | | |
| Limited variety[a] | 2.67 ± .82 | 2.76 ± .91 |
| Food refusal[b] | 1.83 ± .59 | 1.84 ± .61 |

BMIZ, BMI *z*-score

[a] Range (Min-Max): 1.0-4.75 for baseline and 1.0-5.0 for one-year follow-up
[b] Range (Min-Max): 1.0-4.0 for both baseline and one-year follow-up

**Table 4.2.** Bivariate correlations and descriptive statistics for home feeding environment variables at baseline (n = 497)

| | Mean | SD | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. TV in kitchen | 46 (9.5)[a] | | – | – | – | – | – | – | – | – |
| 2. TV in view during meals | 207 (42.6)[a] | | .36* | – | – | – | – | – | – | – |
| 3. TV on during meals | 266 (56.7)[a] | | .48* | .86* | – | – | – | – | – | – |
| 4. Family mealtime routine | 3.74 | .80 | .15 | −.19* | −.29* | – | – | – | – | – |
| 5. CFPQ Monitoring | 4.12 | .94 | −.09 | −.00 | −.16* | .24* | – | – | – | – |
| 6. CFPQ Environment | 3.82 | .61 | −.13 | −.19* | −.23* | .34* | .43* | – | – | – |
| 7. CFPQ Child control | 2.46 | .65 | .00 | .17* | .29* | −.22* | −.16* | −.14* | – | – |
| 8. CFPQ Emotion regulation | 1.50 | .60 | .10 | .08 | .25* | −.09 | −.19* | −.24* | .30* | – |
| 9. CFPQ Balance and variety | 4.29 | .64 | −.03 | −.05 | −.11 | .28* | .34* | .49* | −.13* | −.23* |
| 10. CFPQ Food as reward | 2.17 | .83 | −.02 | .07 | .10 | −.12* | −.13* | −.25* | .14* | .37* |
| 11. CFPQ Involvement | 2.92 | .95 | −.01 | −.15* | −.15* | .20* | .17* | .22* | .01 | −.06 |
| 12. CFPQ Modeling | 3.70 | .88 | .13 | −.07 | −.10 | .39* | .31* | .51* | −.15* | −.13* |
| 13. CFPQ Pressure | 2.54 | .79 | .05 | −.01 | −.00 | .00 | −.03 | −.15* | −.08 | .18* |
| 14. CFPQ Restriction for health | 2.79 | .90 | .06 | .06 | .06 | .01 | .14* | −.12* | .07 | .14* |
| 15. CFPQ Restriction for weight | 1.68 | .55 | −.01 | −.05 | .02 | .02 | .18* | .06 | .04 | .16* |
| 16. CFPQ Teach nutrition | 3.61 | .83 | .06 | −.15* | −.16* | .26* | .28* | .35* | −.06 | −.17* |

CFPQ, Comprehensive Feeding Practices Questionnaire
* $p < .05$
[a] Values for television variables are presented as n (%)

Note: Television variables (TV in kitchen, TV in view during meals, and TV on during meals) were categorical variables coded as 0 = no, 1 = yes. For categorical variables, the value of the correlation coefficient was obtained using polychoric correlation. All other correlation coefficients were obtained from Spearman's rank correlations.

**Table 4.2. (cont.)**

| | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|
| 1. TV in kitchen | – | – | – | – | – | – | – | – |
| 2. TV in view during meals | – | – | – | – | – | – | – | – |
| 3. TV on during meals | – | – | – | – | – | – | – | – |
| 4. Family mealtime routine | – | – | – | – | – | – | – | – |
| 5. CFPQ Monitoring | – | – | – | – | – | – | – | – |
| 6. CFPQ Environment | – | – | – | – | – | – | – | – |
| 7. CFPQ Child control | – | – | – | – | – | – | – | – |
| 8. CFPQ Emotion regulation | – | – | – | – | – | – | – | – |
| 9. CFPQ Balance and variety | – | – | – | – | – | – | – | – |
| 10. CFPQ Food as reward | −.09 | – | – | – | – | – | – | – |
| 11. CFPQ Involvement | .35* | .03 | – | – | – | – | – | – |
| 12. CFPQ Modeling | .65* | −.09 | .34* | – | – | – | – | – |
| 13. CFPQ Pressure | .06 | .31* | .06 | .08 | – | – | – | – |
| 14. CFPQ Restriction for health | .03 | .25* | .02 | .07 | .11* | – | – | – |
| 15. CFPQ Restriction for weight | .01 | .22* | .10* | .12* | .02 | .41* | – | – |
| 16. CFPQ Teach nutrition | .55* | .01 | .56* | .48* | −.02 | .04 | .08 | – |

CFPQ, Comprehensive Feeding Practices Questionnaire
* $p < .05$
[a] Values for television variables are presented as n (%)

Note: Television variables (TV in kitchen, TV in view during meals, and TV on during meals) were categorical variables coded as 0 = no, 1 = yes. For categorical variables, the value of the correlation coefficient was obtained using polychoric correlation. All other correlation coefficients were obtained from Spearman's rank correlations.

**Table 4.3.** Odds ratios (OR) and 95% confidence intervals (CI) for child picky eating behavior at one-year follow-up (n = 326)

| | **Picky eating behavior outcomes at one-year follow-up** | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **Limited variety** | | | | **Food refusals** | | | |
| **Home feeding environment variables at baseline** | **Model 1**[a] | | **Model 2**[b] | | **Model 1**[a] | | **Model 2**[b] | |
| | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** |
| TV in kitchen | 1.90 | .63-5.69 | 1.70 | .52-5.58 | .45 | .12-1.79 | .23 | .04-1.50 |
| TV in view during meals | 2.16* | 1.06-4.39 | .95 | .37-2.45 | 2.53* | 1.25-5.14 | 1.76 | .55-5.66 |
| TV on during meals | 3.31* | 1.47-7.45 | 3.15* | 1.06-9.35 | 1.25 | .61-2.54 | .94 | .28-3.13 |
| Family mealtime routine | .57* | .36-.92 | .58 | .32-1.06 | .73 | .46-1.16 | .80 | .40-1.61 |
| CFPQ Monitoring | .87 | .60-1.26 | .84 | .52-1.35 | 1.20 | .79-1.83 | 1.23 | .68-2.24 |
| CFPQ Environment | .76 | .42-1.39 | .93 | .38-2.26 | .73 | .40-1.33 | .43 | .16-1.15 |
| CFPQ Child control | 1.69 | .93-3.06 | 1.06 | .50-2.24 | 1.47 | .81-2.66 | 3.12* | 1.24-7.83 |
| CFPQ Emotion regulation | 1.31 | .73-2.36 | 1.17 | .56-2.44 | .66 | .36-1.21 | .37* | .15-.90 |
| CFPQ Balance and variety | 1.42 | .80-2.53 | 2.78* | 1.09-7.08 | 1.18 | .67-2.09 | 1.64 | .54-5.02 |
| CFPQ Food reward | 1.07 | .71-1.61 | 1.07 | .61-1.88 | 1.08 | .72-1.63 | 1.27 | .68-2.39 |
| CFPQ Involvement | .94 | .64-1.38 | 1.24 | .73-2.10 | .75 | .51-1.11 | .90 | .47-1.72 |
| CFPQ Modeling | .85 | .58-1.25 | .67 | .35-1.30 | 1.01 | .68-1.49 | 1.53 | .73-3.21 |
| CFPQ Pressure | 1.14 | .74-1.76 | .94 | .56-1.60 | 1.18 | .76-1.83 | 1.44 | .80-2.59 |
| CFPQ Restriction for health | 1.09 | .73-1.61 | .84 | .48-1.48 | 1.03 | .69-1.53 | .53 | .27-1.03 |
| CFPQ Restriction for weight | .99 | .49-1.99 | 1.23 | .51-2.96 | .82 | .42-1.59 | 1.22 | .47-3.23 |
| CFPQ Teach nutrition | .94 | .61-1.44 | .76 | .39-1.48 | .77 | .50-1.18 | .56 | .26-1.23 |

BMIZ, BMI *z*-score; CFPQ, Comprehensive Feeding Practices Questionnaire

* $p < .05$

[a] Model 1 control variables include child age, sex, BMIZ, race/ethnicity, breastfeeding history, parent education, and baseline picky eating behavior

[b] Model 2 includes all control variables from Model 1 and all other variables of the home feeding environment.

# CHAPTER 5:
# Picky Eating in Young Children is Influenced by Genetic Polymorphisms Related to Appetite[5]


## Abstract

**Background:** Picky eating can negatively impact children's growth and development and may be due to low appetite. Individual genetic variations related to appetite may influence picky eating behavior. The objective of this study was to investigate the association between individual single nucleotide polymorphisms (SNPs) in genes related to appetite with picky eating behavior in a cohort of toddlers.

**Methods:** Participants were 231 parents and their 24-month-old children from the STRONG Kids 2 longitudinal birth cohort study. Picky eating was measured using the food fussiness subscale of the Children's Eating Behavior Questionnaire. Length and weight were measured to calculate weight-for-length *z*-scores (WFLZ) and genomic DNA was extracted from the child's saliva. Generalized linear models were used to examine associations between picky eating with ten SNPs in seven appetite-related genes (*DAT1*, *DRD2*, *GHRL*, *LEP*, *LEPR*, *MC4R*, and *PYY*).

**Results:** There was an association between toddlers' picky eating behavior and three individual SNPs: rs40184 in *DAT1*, rs11761556 in *LEP*, and rs1137101 in *LEPR*. Picky eating was not related to WFLZ. There was no relation between picky eating and WFLZ. However, rs2070592 in *PYY* was associated with WFLZ.

**Conclusion:** There is a role for appetite-related SNPs in toddler's picky eating behavior.


[5] To be submitted to *Human Heredity*.

## 5.1 Introduction

Toddlers are at risk for unhealthy food consumption and inadequate nutrient intake (Ahluwalia et al., 2016; Miles & Siega-Riz, 2017). One possibility for these dietary behaviors is picky eating, which is characterized by a limited dietary variety and rejection of familiar and novel foods (Taylor et al., 2015). Picky eating is common in toddlers, with an estimated prevalence of 22% among children two years of age and under (Carruth et al., 2004a; Cole et al., 2017a). For most young children, picky eating is a transient behavior and considered a normal phase of development (Cardona Cano et al., 2016; Nicholls et al., 2001). However, some studies suggest that picky eating remains relatively stable through childhood and early adulthood (Ashcroft et al., 2008; Mascola et al., 2010; Van Tine et al., 2017).

Previous work has shown picky eating can have adverse consequences on children's weight and development, including slower growth, lower body mass index, lower fat-free mass, and a higher risk of becoming underweight (Antoniou et al., 2016; de Barse et al., 2015; Dubois et al., 2007a; Ekstein et al., 2010; Kwon et al., 2017; Xue et al., 2015). Picky eating is also related to unhealthy dietary patterns and nutrient deficiencies (Taylor et al., 2016; van der Horst et al., 2016; Volger et al., 2017). Compared to non-picky eaters, picky eaters eat slower (Cardona Cano et al., 2015; Fildes et al., 2015; Mascola et al., 2010; Reau et al., 1996), have a lower enjoyment of food (Fildes et al., 2015; Sleddens et al., 2008; van der Horst, 2012), prefer to drink instead of eat what was served at the meal, have a lower acceptance of common foods, and are less willing to come to the table at mealtime (Boquin et al., 2014b). Children who are picky eaters may have a lower desire to eat (Webber et al., 2009) and lower appetite (Wright et al., 2006; Wright et al., 2007). However, additional research is needed to determine if individual differences in appetite contribute to variation in picky eating behavior.

Genetics play a significant role in the etiology of picky eating, with heritability estimates from twin studies ranging from 72% to 78% in children (Cooke et al., 2007; Faith et al., 2013; Fildes et al., 2016). Differences in child appetitive traits are also related to genetics, with heritability estimates from twin studies ranging from 63% to 84% for satiety responsiveness and slowness in eating, 53% to 75% for food responsiveness and enjoyment of food, and 56% to 89% for not eating enough (Carnell et al., 2008; Dubois et al., 2013). In a recent study, Monnereau et al. (2017) tested the relation between eating behavior and genetic variants associated with body mass index, including *BDNF*, *FTO*, and *MC4R*, in a sample of preschool-aged children. They found an association between individual genetic variants in *BDNF* and satiety responsiveness; however, they did not find an association between any of the investigated genetic variants and food fussiness. Genetic variants related to appetite control may influence children's desire to eat and their enjoyment and rejection of food, which could impact picky eating behavior. Since picky eating behavior emerges in toddlerhood and peaks in preschool years (Cardona Cano et al., 2015), additional research is needed to investigate genetic influences that may contribute to variation in picky eating behavior in younger children.

The primary aim of the present study was to identify the influence of individual genetic variations on picky eating behavior in a sample of non-Hispanic white toddlers. Specifically, we determined whether single nucleotide polymorphisms (SNPs) in genes related to appetite were associated with parent-reported food fussiness. We also tested the relationship between genetic variation and child weight-for-length *z*-scores (WFLZ). We hypothesized that individual genetic variations in genes involved in appetite would be associated with child picky eating behavior and WFLZ.

## 5.2 Methods

### Participants

Participants were drawn from the Synergistic Theory Research Obesity and Nutrition Group (STRONG) Kids 2 (SK2) birth cohort study, which aims to identify the interaction between biological and environmental factors in predicting eating habits and obesity risk in young children. Women were recruited in their third trimester of pregnancy from birthing classes and healthcare facilities in east-central Illinois. To control the effects of genetic ancestry, analysis was restricted to non-Hispanic white children. A subsample of non-Hispanic white children ($n$ = 231) for whom complete genetic data were available were included in the study. Parents provided written consent for their child's participation in the study and completed an online or paper survey at six weeks and 24 months. This research was reviewed and approved by the Institutional Review Board at the University of Illinois at Urbana-Champaign.

### Measures

#### *Picky Eating Behavior*

Picky eating behavior was measured using the food fussiness subscale of the Children's Eating Behavior Questionnaire (CEBQ), a validated and widely used tool for assessing differences in children's eating behaviors (Wardle et al., 2001). Parents filled out the CEBQ when the children were 24 months old. The food fussiness scale consists of six items (e.g., "My child refuses new foods at first") in which parents rate their child's picky eating behavior and selectivity regarding the range of accepted and rejected foods using a Likert scale from 1 (never) to 5 (always). A total score for the food fussiness subscale was calculated by taking the mean of item scores; high scores on the subscale represented higher levels of picky eating behavior. Cronbach's alpha for this subscale was 0.90.

### *Anthropometric Measures*

Children's weight (kg) and length (cm) at 24 months were measured at the child's home by trained research personnel. Length was obtained by measuring the distance between the child's head and feet while lying flat on a scale (SECA, Model 728, Hamburg, Germany) and weight was measured using an electronic remote display scale (Health-O-meter, Model 349KLX, McCook, IL, USA). Three measurements were taken at each visit and the average of the three measurements was recorded. Age- and sex-specific WFLZ at 24 months was calculated using the 2006 World Health Organization (WHO) growth charts (Grummer-Strawn et al., 2010).

### *Candidate Gene and SNP Selection*

Identification of candidate genes was based on a review of published literature regarding the gene's putative role in appetite. Previous research has associated energy intake and eating behavior with genes related to appetite control, including *BDNF*, *FTO*, *GHRL*, *LEP*, *LEPR*, *MC4R*, and *PYY* (Arija et al., 2010; Cecil et al., 2008; Cole et al., 2010; de Krom et al., 2007; Ho-Urriola et al., 2014; Obregón et al., 2017; Valladares et al., 2010; Wardle et al., 2008; Wardle et al., 2009). Variation in genes related to reward function may also influence appetitive traits, thus dopaminergic genes (*COMT*, *DAT1*, *DRD2*, and *DRD4*) were also included (Obregon et al., 2017; Stice et al., 2010; Volkow et al., 2011; Wang et al., 2001). SNP selection was performed using a systematic procedure based on their location near or within each gene of interest. A total of 13 candidate SNPs were identified: rs6265 (*BDNF*), rs4680 (*COMT*), rs40184 (*DAT1*), rs1800497 and rs2283265 (*DRD2*), rs1800955 (*DRD4*), rs8057044 (*FTO*), rs27647 (*GHRL*), rs1349419 and rs11761556 (*LEP*), rs1137101 (*LEPR*), rs17782313 (*MC4R*), and rs2070592 (*PYY*).

### *DNA Extraction and Genotyping*

DNA was isolated from saliva samples collected by trained research personnel when the child was six weeks of age or, for a small subgroup with low saliva yield, at 24 months of age. Genomic DNA extraction was conducted using the Oragene DNA protocol for the manual purification of DNA from saliva samples. The Functional Genomic Unit of the W.M. Keck Center at the University of Illinois performed sample pre-amplification and genotyping. Genotypes were called by two researchers at a minimum of 95% confidence using Fluidigm® Genetic Analysis (Version 4.1.2, San Francisco, CA, USA). Samples that failed genotyping for any of the investigated markers were excluded (n=27), leaving a final sample size of 231 participants.

### Statistical Analysis

All statistical analyses were conducted using Statistical Analysis Software (Version 9.4, SAS Institute, Cary, NC, USA). Statistical significance was determined at $p < 0.05$. Sample characteristics are expressed as means ± standard deviations for continuous data, and frequencies are expressed as percentages for categorical data. Deviation from Hardy-Weinberg equilibrium (HWE) was evaluated using Chi-square test. SNPs were included in analyses if the genotype call rate confidence was > 95%, HWE *p*-value was > 0.05, and the minor allele frequency (MAF) was > 5% (as reported for CEU in HapMap, http://hapmap.ncbi.nlm.nih.gov/). Three SNPs (*BDNF* rs6265, *COMT* rs4680, and *FTO* rs8057044) were in extreme HWE deviation ($p < 0.05$) and *DRD4* rs1800955 had a call rate confidence level < 95%. Therefore, these four SNPs were not included in further analysis. Linkage disequilibrium (LD) analyses was performed on *DRD2* and *LEP*. The two *DRD2* variants were in strong LD ($R^2 = 0.68$, $D^2 = 0.98$). Thus, results are

presented for rs1800497 only. The *LEP* variants were not in LD; thus, results are presented for both rs1349419 and rs11761556.

Independent-sample *t* test was used to evaluate differences in picky eating by child WFLZ. Generalized linear models were used to determine the association between individual SNPs with child picky eating and WFLZ. Each SNP was tested for allelic associations with picky eating and WFLZ under additive, dominant, and recessive models. The picky eating model was adjusted for child sex and parent education level (two levels: college graduate/post-graduate versus grade school/high school/some college). The WFLZ model was already sex and age adjusted, thus, was only adjusted for parent education level. To identify possible sex-specific associations with picky eating, individual SNPs were also analyzed for evidence of gene by sex interaction. We also identified possible gene by parent education interactions.

## 5.3 Results

### Study Characteristics

**Table 5.1** provides a summary of the eight candidate SNPs that were analyzed: rs40184 (*DAT1*); rs1800497 (*DRD2*); rs27647 (*GHRL*); rs1349419 and rs11761556 (*LEP*); rs1137101 (*LEPR*); rs17782313 (*MC4R*); and rs2070592 (*PYY*). All SNPs were in HWE according to Chi-square analysis ($p > 0.05$). At age 24 months, the child sample was 53% female. Mean child WLFZ was 0.83 ± 1.07 (95% confidence interval, 0.68-0.97). The majority of parents had a college degree (77%) and were employed (78%). On average, children's food fussiness score was 2.67 ± 0.74 (95% confidence interval, 2.57-2.76). Food fussiness was not related to child sex or child WFLZ. There was no significant difference in WFLZ between boys and girls.

**Association between Genotype and Picky Eating**

**Table 5.2** contains the associations between genotype and child picky eating, as measured by food fussiness scores. There was a significant difference in picky eating according to genotype for *DAT1*, *LEP*, and *LEPR* while controlling for child sex and parent education. For *DAT1*-rs40184, children with the A/G genotype had lower food fussiness scores (2.43 ± 0.11) compared to homozygotes (A/A: 2.88 ± 0.23; G/G: 2.71 ± 0.08). For *LEP*-rs11761556, C/C homozygotes had lower food fussiness scores compared to A-allele carriers (2.35 ± 0.11 vs. 2.69 ± 0.07, $p = 0.007$). For *LEPR*-rs1137101, G/G homozygotes had lower food fussiness scores compared A-allele carriers (2.35 ± 0.14 vs. 2.69 ± 0.07, $p = 0.02$).

A significant sex-by-genotype interaction was observed for food fussiness involving *LEP*. For rs11761556, males with the C/C genotype had lower food fussiness scores compared to A-allele carriers (2.15 ± 0.16 vs. 2.86 ± 0.09, $p = .0001$). For rs1349419, males with the A/A genotype had lower food fussiness scores compared to G-allele carriers (2.35 ± 0.16 vs. 2.76 ± 0.10, $p = .012$).

A significant sex-by-education interaction was observed for food fussiness involving *DRD2*-rs1800497. Among children whose parents did not have a college degree ($n = 52$), those with the C/C genotype had lower food fussiness scores compared to T-allele carriers (2.24 ± 0.14 vs. 2.96 ± 0.21, $p = .006$).

**Association between Genotype and WFLZ**

**Table 5.3** contains the associations between genotype and child WFLZ. There was a significant association between individual genetic variation in *PYY* and WFLZ. *PYY*-rs2070592 G-allele carriers had lower WFLZ compared to A/A homozygotes (0.84 ± 0.12 vs. 1.28 ± 0.14, $p = 0.01$).

## 5.4 Discussion

To the best of our knowledge, this is the first study to investigate the association between individual genetic variations related to appetite and picky eating behavior in a sample of toddlers. Of the seven candidate genes investigated, an association was observed between picky eating and genetic variants in *DAT1* (dopamine transporter 1), *LEP* (leptin), and *LEPR* (leptin receptor). There was no relation between picky eating and WFLZ. However, genetic variation in *PYY* (peptide YY) was associated with WFLZ.

Picky eating has been suggested to be associated with having lower appetite, food intake, and desire to eat (van der Horst et al., 2016; Webber et al., 2009; Wright et al., 2006; Wright et al., 2007). Leptin plays a key role in appetite regulation and energy balance; leptin is an appetite-suppressing hormone produced by adipose tissue (Klok et al., 2007). Variations in *LEP* and *LEPR* in children have been implicated in obesity-related phenotypes, parent-reported nutritional risk, and emotional eating (Michels et al., 2017; Persaud et al., 2017). In support of our hypothesis, findings from this study suggest that picky eaters may have reduced appetite based on genetic differences. Further, variations in *LEP* were associated with differences in picky eating behaviors among boys. Previous studies have found that boys are pickier eaters compared to girls (Powell et al., 2011; Sleddens et al., 2008).

The *DAT1* gene (also known as *SLC6A3*) encodes DAT1 proteins involved in the reuptake of dopamine from neural synapses and these proteins are densely populated in the mesolimbic reward pathway (Doucette-Stamm et al., 1995). Low dopamine availability in the reward pathway could increase children's vulnerability to conduct problems in adverse environments (Gatzke-Kopp, 2011). Previous studies in children have associated genetic variation in *DAT1* with hyperactivity, sensation seeking, and disruptive behavior problems

(Caylak, 2012; Davies et al., 2015; Rommelse et al., 2008). In the present study, heterozygotes for *DAT1*-rs40184 were the least picky eaters, demonstrating heterozygote advantage (Sellis et al., 2011). Children who are more sensation seeking may be more willing to try new foods (and thus, are less food neophobic).

Similar to other studies in young children, we did not identify a relation between adiposity and picky eating (Quah et al., 2015; Wright et al., 2007). However, homozygosity for the *PYY*-rs2070592 A allele was associated with higher adiposity, which aligns with the findings from a previous study in which individual genetic variation in *PYY* was associated with childhood obesity (Siddiq et al., 2007). *PYY* encodes the gastrointestinal hormone Peptide YY, which is released by gastrointestinal cells after a meal to reduce appetite and food intake, and slow gastric emptying (Batterham & Bloom, 2003; le Roux & Bloom, 2005). Lower levels of PYY in response to a meal have been associated with obesity in children (Nguo et al., 2016; Roth et al., 2005).

This study has several limitations. First, the small sample size may limit statistical power to identify some of the relations between individual genetic variations and picky eating. Second, although the food fussiness subscale is based on validated questionnaires, picky eating was assessed using parent-report instead of objective measurements. Third, the sample was restricted to non-Hispanic white children, thus potentially reducing the generalizability of our findings. Finally, other alleles commonly implicated in the dopaminergic system were not included. We established standard procedure to genotype the *DRD4* 7-repeat allele; however, we were unable to include this genetic variant in the study due to limited participant DNA samples.

## 5.5 Conclusion

In conclusion, this investigation provides evidence that variants in appetite-related genes may play a role in picky eating behavior in toddlers. These findings may explain differences in appetitive behavior between picky eaters and non-picky eaters, such as a lower drive to eat and a longer mealtime duration. Further work is necessary to understand the influence of appetite-related genes on toddler's picky eating behavior and weight trajectory.

## 5.6 Tables

**Table 5.1.** Characteristics of selected single nucleotide polymorphisms (SNPs)

| Gene | SNP | Basic Information | | SK2 Cohort | |
|---|---|---|---|---|---|
| | | **Chromosome: position, kb** | **Reported MAF**[a] | **Detected MAF** | **HWE *p* value** |
| *DAT1* | rs40184 | 5: 1394962 | A 0.46 | A 0.21 | 0.17 |
| *DRD2* | rs1800497 | 11: 113400106 | T 0.19 | T 0.19 | 0.17 |
| *GHRL* | rs27647 | 3: 10290784 | C 0.34 | C 0.39 | 0.63 |
| *LEP* | rs1349419 | 7: 128237160 | A 0.39 | G 0.48 | 0.34 |
| | rs11761556 | 7: 128257016 | C 0.44 | A 0.49 | 0.50 |
| *LEPR* | rs1137101 | 1: 65592830 | G 0.47 | G 0.43 | 0.76 |
| *MC4R* | rs17782313 | 18: 60183864 | C 0.30 | C 0.49 | 0.29 |
| *PYY* | rs2070592 | 17: 43953963 | G 0.28 | G 0.37 | 0.89 |

HWE = Hardy-Weinberg equilibrium; MAF = Minor Allele Frequency; SK2= STRONG Kids 2.

[a] Caucasian population (HAPMAP).

**Table 5.2.** Selected single nucleotide polymorphisms (SNPs) and associations with picky eating

| SNP | Genotype | N (%) | Model[a], p-value | | | |
|---|---|---|---|---|---|---|
| | | | LSMeans ± se | Additive | Dominant | Recessive |
| rs40184 | AA | 11 | 2.875 ± 0.230 | **0.0441** | 0.2813 | 0.0833 |
| | AG | 53 | 2.426 ± 0.108 | | | |
| | GG | 113 | 2.708 ± 0.079 | | | |
| rs1800497 | CC | 114 | 2.603 ± 0.080 | 0.9820 | 0.8571 | 0.9989 |
| | CT | 47 | 2.578 ± 0.115 | | | |
| | TT | 9 | 2.595 ± 0.255 | | | |
| rs27647 | CC | 28 | 2.669 ± 0.144 | 0.1591 | 0.4734 | 0.0550 |
| | CT | 79 | 2.657 ± 0.088 | | | |
| | TT | 65 | 2.443 ± 0.094 | | | |
| rs1349419 | AA | 51 | 2.492 ± 0.107 | 0.2402 | 0.3289 | 0.1006 |
| | AG | 83 | 2.544 ± 0.085 | | | |
| | GG | 45 | 2.728 ± 0.113 | | | |
| rs11761556 | AA | 44 | 2.716 ± 0.114 | **0.0235** | 0.2000 | **0.0065** |
| | AC | 82 | 2.669 ± 0.086 | | | |
| | CC | 47 | 2.348 ± 0.109 | | | |
| rs1137101 | AA | 55 | 2.586 ± 0.106 | **0.0263** | **0.0199** | 0.6031 |
| | AG | 88 | 2.762 ± 0.087 | | | |
| | GG | 32 | 2.350 ± 0.136 | | | |
| rs17782313 | CC | 45 | 2.633 ± 0.117 | 0.7799 | 0.7080 | 0.6770 |
| | CT | 79 | 2.553 ± 0.093 | | | |
| | TT | 48 | 2.635 ± 0.115 | | | |
| rs2070592 | AA | 68 | 2.703 ± 0.097 | 0.2791 | 0.1907 | 0.7870 |
| | AG | 79 | 2.510 ± 0.093 | | | |
| | GG | 24 | 2.672 ± 0.158 | | | |

Bolded *p* values are significant at $\alpha = 0.05$. [a] Adjusted for child sex and parent education level

**Table 5.3.** Selected single nucleotide polymorphisms (SNPs) and associations with weight-for-length *z*-scores (WFLZ)

| SNP | Genotype | N (%) | Model[a], p-value | | | |
|---|---|---|---|---|---|---|
| | | | LSMeans ± se | Additive | Dominant | Recessive |
| rs40184 | AA | 11 | 1.591 ± 0.340 | 0.1564 | 0.0541 | 0.5507 |
| | AG | 53 | 0.905 ± 0.175 | | | |
| | GG | 113 | 0.928 ± 0.123 | | | |
| rs1800497 | CC | 114 | 0.967 ± 0.117 | 0.7685 | 0.5213 | 0.5901 |
| | CT | 47 | 1.060 ± 0.168 | | | |
| | TT | 9 | 1.198 ± 0.382 | | | |
| rs27647 | CC | 28 | 0.934 ± 0.224 | 0.2006 | 0.9451 | 0.0861 |
| | CT | 79 | 0.806 ± 0.136 | | | |
| | TT | 65 | 1.137 ± 0.142 | | | |
| rs1349419 | AA | 51 | 0.788 ± 0.171 | 0.3413 | 0.1531 | 0.8359 |
| | AG | 83 | 1.089 ± 0.138 | | | |
| | GG | 45 | 1.015 ± 0.186 | | | |
| rs11761556 | AA | 44 | 1.088 ± 0.178 | 0.5955 | 0.5036 | 0.3370 |
| | AC | 82 | 1.017 ± 0.134 | | | |
| | CC | 47 | 0.865 ± 0.160 | | | |
| rs1137101 | AA | 55 | 1.071 ± 0.155 | 0.2983 | 0.5329 | 0.1205 |
| | AG | 88 | 1.047 ± 0.131 | | | |
| | GG | 32 | 0.717 ± 0.202 | | | |
| rs17782313 | CC | 45 | 0.931 ± 0.173 | 0.2064 | 0.7021 | 0.0795 |
| | CT | 79 | 0.869 ± 0.135 | | | |
| | TT | 48 | 1.215 ± 0.165 | | | |
| rs2070592 | AA | 68 | 1.279 ± 0.143 | **0.0366** | 0.1903 | **0.0114** |
| | AG | 79 | 0.873 ± 0.134 | | | |
| | GG | 24 | 0.752 ± 0.219 | | | |

Bolded *p* values are significant at $\alpha = 0.05$. [a] Adjusted for parent education level

# CHAPTER 6:
# Impact of Reported and Observed Parental Feeding Responsiveness on Toddlers' Picky Eating Behavior[6]


## Abstract

**Background:** Picky eating behavior is prevalent among toddlers and may be related to parental feeding responsiveness during mealtimes. Research is needed in the naturalistic home environment to better understand the influence of parental feeding responsiveness on child picky eating behavior. The objective of this study was to examine associations between reported and observed responsive feeding practices with picky eating behavior in a sample of toddlers.

**Methods:** Participants were parents and their 18- to 35-month-old children (n = 91) from the STRONG Kids 2 (SK2) study. Parents responded to survey questions on their feeding practices and child's eating behavior. Families were observed during a typical family dinner in the naturalistic home environment. Videos were coded for parents' observed feeding practices. Associations were assessed with Spearman's correlations and hierarchical regressions.

**Results:** Parent-reported nonresponsive feeding practices were predictive of higher toddler picky eating, supporting the hypothesis that responsive feeding is inversely associated with picky eating behavior in young children. However, there was no relation between observed feeding practices and picky eating.

**Conclusions:** Nonresponsive feeding practices may negatively influence the development of healthful eating behavior in young children. Additional observational research is needed to better understand the relation between responsive feeding practices and picky eating.


[6] To be submitted to *Appetite*.

## 6.1 Introduction

The first two years of life are a sensitive period during which children learn and develop food preferences and dietary patterns (Birch & Doub, 2014; Cashdan, 1994). A common behavior among toddlers is picky eating (Carruth et al., 2004a), which is generally characterized as the acceptance of a limited variety of familiar or novel foods (Taylor et al., 2015). Although picky eating is considered a normal mealtime behavior for many healthy young children (Crist & Napier-Phillips, 2001), it has been linked with health-related issues and behaviors, including functional constipation (Tharner et al., 2015), tactile defensiveness (Smith et al., 2005), and sleep problems (Hafstad et al., 2013b). Additionally, picky eating is a frequently reported source of stress and anxiety for parents and caregivers (Goh & Jacob, 2012; Trofholz et al., 2017), who may adapt certain feeding strategies to encourage children to eat (Johnson et al., 2015; Luchini et al., 2017a; Rubio & Rigal, 2017). To promote healthful eating behavior, research is needed to better understand the impact of parental feeding behavior on children's picky eating.

Parental feeding responsiveness is the extent to which parents foster child intake self-regulation and recognition of internal satiety and hunger cues by exhibiting warmth and attunement around meals (Black & Aboud, 2011). In a responsive feeding environment, parents respond to the child in a prompt, developmentally appropriate, and emotionally supportive manner, and provide pleasant and structured mealtimes with few distractions (Black & Aboud, 2011; DiSantis et al., 2011; Eneli et al., 2008). Previous studies have shown that responsive feeding practices, such as providing family meals, are negatively associated with picky eating in children (Brown & Lee, 2015; Cassells et al., 2014; Finnane et al., 2017; Jansen et al., 2014). However, these studies primarily assessed caregiver feeding practices using unidirectional parent self-report measures, which could be subject to recall biases or random responding.

Observational approaches, particularly observations in the naturalistic home environment, are a valuable tool for collecting information on parent-child interactions (Gardner, 2000). By capturing verbal and nonverbal contexts, observational methods can provide insight on parent and child behavior that may not be evident from self-reported measures. From the feeding standpoint, observational measures provide a different perspective on how parents interact with children during mealtimes and information that may diverge from reported feeding practices (Hughes et al., 2013; Moens et al., 2007). Yet, few studies have utilized observational approaches in the investigation of parent-child mealtime interactions, particularly among younger children (Bergmeier et al., 2015a). In a recent observational study, observed parent feeding practices were better predictors of children's observed food refusals compared to parents' reported feeding practices (Fries et al., 2017). Additional observational work in the naturalistic home environment is needed to better understand the influence of parent feeding practices on child picky eating.

Using an observational approach to measure parent feeding practices during a typical family dinner in the naturalistic home setting, the objective of this study was to examine the impact of parental feeding responsiveness on picky eating behavior in a sample of toddlers. The first aim was to examine associations between reported and observed parent feeding practices. The second aim was to evaluate relations between reported and observed parental feeding responsiveness with child picky eating behavior. Based on existing literature on responsive feeding, we hypothesized that responsive feeding would be inversely associated with picky eating behavior in children. Additionally, we hypothesized that parents' observed feeding responsiveness would be more strongly associated with children's picky eating behavior than parents' reported feeding responsiveness.

## 6.2 Methods

### Participants

Primary caregivers and their children (n = 110) in east-central Illinois were recruited between 2015 and 2017 as part of the STRONG Kids 2 (SK2) longitudinal birth cohort study, which is investigating the relation among genetic, child, and family factors in predicting child health outcomes. As part of SK2, parents also completed a comprehensive survey online or on paper that included questions on demographics, feeding practices, and their child's eating behavior. A total of 91 parent-child dyads with complete observational and questionnaire data and were used for subsequent analysis. This research was reviewed and approved by the Institutional Review Board at the University of Illinois at Urbana-Champaign and met all requirements for ethical conduct for research with human subjects.

### Procedure

Parents were invited via telephone or email to participate in a study involving a single home observation of their dinner meal. Using a standardized script, researchers explained that the purpose of the study was to get more information about the many ways in which families with very young children manage mealtimes and routines. Families were instructed to act as they normally would during a typical mealtime. All parents provided written informed consent at the beginning of the home observation. Trained researchers arrived 30-45 minutes before the pre-arranged dinnertime to set-up the digital video camera and acclimate children to having a camera in the room. Video cameras were set up to allow optimal observation of the target child during the mealtime. Once families were ready to start dinner, the video camera was turned on to record the meal, and researchers left the home until the meal was over to minimize any alterations in participant behavior. Families were compensated after the home observation.

## Measures

### *Child Picky Eating Behavior*

Child picky eating behavior was measured when the child was 24 months using the six-item food fussiness subscale of the Child Eating Behavior Questionnaire (CEBQ) (Wardle et al., 2001). Parents were asked to rate their child's selectivity regarding the range of accepted and rejected food using a 5-point Likert-scale from 1 (Never) to 5 (Always). Sample items included the reverse-coded questions "My child enjoys a wide variety of foods" and "My child is interested in tasting food he/she hasn't tasted before". In the current sample, high internal consistency was found for the food fussiness subscale (Cronbach's $\alpha = .90$). Higher mean food fussiness scores indicated more picky eating behavior.

### *Parent-Reported Feeding Styles and Feeding Practices*

The Caregiver's Feeding Styles Questionnaire (CFSQ) was used to measure parent-reported feeding styles when the child was 18 months old (Hughes et al., 2005; Hughes et al., 2011). The CFSQ classifies the overall feeding pattern of parents based on dimensions of responsiveness and demandingness, which are derived from 19 feeding directives (seven child-centered and 12 parent-centered) measured on a 5-point Likert scale from 1 (Never) to 5 (Always). Child-centered feeding directives (or responsive feeding practices) promote child autonomy during mealtimes. Parent-centered feeding directives (or nonresponsive feeding practices) control the child's eating through external pressure. Based on median splits and cross-classification of high and low scores of responsiveness and demandingness, four feeding styles were identified: authoritative (high on responsiveness and demandingness), authoritarian (low responsiveness and high demandingness), indulgent (high responsiveness and low demandingness), and uninvolved (low on responsiveness and demandingness). In the current

sample, the median for responsiveness was 1.36 and for demandingness it was 2.26. To investigate responsive and nonresponsive feeding practices, items from the CFSQ were also investigated as responsive and nonresponsive subscales. In the current sample, Cronbach's α for was .61 for the seven responsive items and .86 for the 12 nonresponsive items.

***Observed Parent Feeding Practices***

Observed parent feeding practices were measured using a 19-item measure adapted from the Feeding Behavior Coding System (FBCS), an observational checklist of the CFSQ that documents specific feeding practices exhibited by parents and their frequency of occurrence (Hughes et al., 2007; Hughes et al., 2011). Feeding practices were divided into responsive and nonresponsive feeding practices based on their congruence with child- and parent-centered feeding directives in the CFSQ. Sample responsive feeding practices included asking the child questions about the food, helping the child to eat, and making positive comments about the food. Sample nonresponsive feeding practices included spoon-feeding and telling the child to eat. Behavioral codes were scored by averaging behaviors for responsive feeding practices and nonresponsive feeding practices.

To account for variation in mealtime duration, mealtime start was defined as the time at which food was placed in front of the child. If food was already in front of child when the camera was turned on, then the start of the mealtime was defined as the time at which the mealtime recording began. The end of the mealtime was defined as the time at which food was taken away from the child, or at 20 minutes (whichever occurred first). Twenty minutes was selected as the maximum assessment period because it is considered an appropriate meal length for young children and has been shown to be a sufficient amount of time to assess feeding interactions in a single observation (Sanders et al., 1993; Turner et al., 1994). Additionally,

research suggests that mealtime duration does not differ between picky eaters and non-picky eaters (Adamson et al., 2015).

Three researchers received extensive in-person training prior to coding parent feeding practices. A comprehensive coding manual was prepared consisting of definitions and detailed examples of each responsive and nonresponsive feeding practice. Mealtime videos were coded using INTERACT professional software for observational data (Version 16, Mangold International, Arnstorf, Germany). To establish inter-rater reliability, 25% of the mealtime videos were selected randomly and coded independently by two coders. Reliability analyses were conducted using Statistical Package for the Social Sciences (Version 24, SPSS Inc., Armonk, NY). Intra-class correlation coefficients between coders ranged from 80% to 95% for the 19 observed feeding practices.

**Statistical Analyses**

Other than reliability analyses for observed feeding practices, all statistical analyses were performed using Statistical Analysis Software (Version 9.4, SAS Institute Inc., Cary, NC). Statistical significance level was defined at $p < .05$. Distributions of the data were first examined through visual inspection of histograms and skewness and kurtosis statistics. The total frequencies of observed parent feeding practices were examined to identify practices that occurred too infrequently for analysis. There were 11 feeding practices that occurred an average of less than .5 times per meal: arranging the food to make it more interesting, reasoning with the child, physically struggling with the child, suggesting the child to eat, hurrying the child to eat, begging the child to eat, promising a food reward, promising a reward other than food, threatening food punishment, threatens other punishment, and making negative comments about the food. These feeding practices were not included in statistical analyses (Hughes et al., 2011).

Spearman's correlations were conducted to test associations between reported and observed feeding practices. Independent sample t-tests and analysis of variance were conducted to examine differences in food fussiness scores by child age, gender, race/ethnicity, parent age, education level, employment status, length of mealtime, and the number of people present at the meal. Least significant difference tests were used to determine the difference in observed feeding practices by feeding style. Observed feeding practices were positively skewed, thus natural log transformation was applied to each feeding practice to improve the distribution prior to conducting least significant difference tests. Hierarchical regression was used to test the relation between reported and observed feeding as predictors of child food fussiness. The covariates parental education level and length of the mealtime were entered at step 1, observed responsive and nonresponsive feeding practices were entered at step 2, and reported responsive and nonresponsive feeding practices were entered at step 3, and reported feeding style was entered at step 4.

### 6.3 Results

Characteristics of the 91 parent-child dyads are presented in **Table 6.1**. At the time of the home mealtime observation, children participating in the study ranged from 18 to 34.9 months and were equally distributed across child gender. On average, meal length was 17.4 minutes (SD = 3.9; Range = 6 to 25) with 3.4 people present at the meal (SD = 1.1; Range = 2 to 9). The mean of children's food fussiness scores was 2.7 (SD = .8, Range = 1 to 4.8) and did not vary according to child age, gender, race/ethnicity, parent age, education level, employment status, meal length, or the number of people present at the mealtime. In the current sample, parents' feeding styles were classified as follows: authoritarian ($n$ = 30), authoritative ($n$ = 14), indulgent

($n$ = 27), and uninvolved ($n$ = 20). Child food fussiness was unrelated to feeding style and observed feeding practices.

**Table 6.2** presents the means, standard deviations, and minimum and maximum values for observed parent feeding practices. In general, responsive feeding practices occurred more frequently compared to nonresponsive feeding practices. Total feeding practices demonstrated wide variability and ranged from 5 to 102 (mean ± SD = 40.1 ± 21.3). The most commonly observed responsive feeding practices among parents were asking the child questions about the food and allowing the child to choose from available options during the mealtime. Spoon feeding and telling the child to eat were the most commonly observed nonresponsive feeding practices.

**Table 6.3** contains the Spearman correlations among reported and observed parent feeding practices. The following maternal reported behaviors were positively associated with their observed counterparts: praise/approval ($r$ = .21, $p$ < .05), spoon feeding ($r$ = .21, $p$ < .05), and telling the child to eat ($r$ = .25, $p$ < .05). For both reported and observed measures, praising/approving the child during mealtime was positively associated with providing positive comments, telling the child to eat, and disapproving/scolding the child. **Table 6.4** presents the feeding style differences on observed feeding practices. Indulgent parents were least likely to tell their child to eat compared to authoritative, authoritarian, and uninvolved parents.

**Table 6.5** presents the regression results predicting children's food fussiness. The overall $F$ value of 3.78 ($df$ = 7, 80), with a multiple $R$ of .43, was significant at the .01 level. After controlling for mealtime length and parent education level, observed feeding practices were not shown to account for a significant amount of additional variance in food fussiness (the $R^2$ change was not significant with adjusted $R^2$ = .03 and $F$(4, 83) = 1.45). After accounting for covariates and observed feeding practices, reported feeding practices was shown to account for an

additional 0.11 amount of variance in food fussiness ($F\Delta$ (2, 80) = 14.65, $p < .001$), indicating the addition of reported feeding practices significantly improved $R^2$. Reported nonresponsive feeding practices was a positive predictor of child food fussiness. When adding feeding style to the model, reported nonresponsive feeding practices continued to be significantly associated with child food fussiness.

### 6.4 Discussion

To the best of our knowledge, this is the first study to include both self-reported and observed measures of parental feeding responsiveness during a home-based mealtime observation. With respect to child picky eating behavior, parent-reported nonresponsive feeding practices were predictive of higher toddler food fussiness scores. This finding supports the hypothesis that responsive feeding is inversely associated with picky eating behavior in young children. However, contrary to the hypothesis, parent-reported responsive feeding was a better predictor of children's picky eating behavior than observed responsive feeding.

Only three feeding practices were significantly associated between reported and observed measures, which aligns previous findings showing that parent-reported feeding practices are not always significantly associated with observed feeding practices (Bergmeier et al., 2015b; Haycraft & Blissett, 2008; Lewis & Worobey, 2011). There are several potential reasons for the lack of congruency between reported and observed feeding practices. In the present study, some mealtimes were conducted with only the mother present at the mealtime. Previous research suggests that fathers and other influential caregivers, such as grandparents and older siblings, may alter feeding interactions (Blissett et al., 2006; Farrow, 2014; Haycraft & Blissett, 2008; Jiang et al., 2007; Payne et al., 2011). In addition, parents may have altered their feeding

practices when being observed in the home. Multiple home observations may minimize differences in reported and observed feeding practices. Praising/approving the child (a responsive feeding practice) was positively associated with telling the child to eat and disapproving/scolding the child, both of which are non-responsive feeding practices. This could be a reflection of parents' overall level of communication during a meal – parents that talk more in general may have a higher tendency to exhibit both verbally responsive and non-responsive feeding practices. This suggests that coding procedures may need to incorporate additional measures on the quality of parent input (e.g., positive versus negative tone) during the meal, as this could be driving child eating behavior rather than specific feeding practices.

There was no association between parent feeding style and picky eating. However, compared to other feeding styles, indulgent parents were least likely to tell their child to eat (a nonresponsive feeding practice). This finding aligns that of previous research, which found that parents with an indulgent feeding were observed to be significantly lower on verbal directive feeding practices compared to parents of other feeding styles (Hughes et al., 2011). There was no association between observed responsive feeding and child picky eating behavior. Unlike Fries et al. (2017), reported parent-reported feeding practices in this study were better predictors of children's food fussiness compared to parents' observed feeding practices. One potential reason for this finding is that picky eating behavior was measured based on parental perception. Observed parental feeding practices may be more predictive of observed child eating behavior, which is a measure this study did not capture. Another reason is that observed responsive feeding may be a better predictor of child picky eating behavior later in childhood. Reported measures of parent feeding practices may be capturing unique aspects of feeding responsiveness that are not

captured through observation. However, the opposite is also true. Future studies investigating parent feeding behavior may benefit from utilizing both observational and reported measures.

There was a wide range in the number of feeding practices used by parents during the mealtime. In the present study, parents tended to ask questions about the food or allow the child to choose from available food options in order to get the child to eat. Asking children questions during the mealtime encourages their communication about food and provides opportunities to expand their vocabulary (Snow & Beals, 2006). Allowing children to choose from available food options supports the development of self-regulation of energy intake (Johnson & Taylor-Holloway, 2006; Tan & Holub, 2011). In the present study, parents also frequently spoon-fed their children. According to a study on infant and toddler feeding behaviors, 80% of children were self-feeding with spoons by age 24 months (Carruth & Skinner, 2002). An alternative approach to traditional spoon-feeding is baby-led weaning, which involves presenting finger foods to young children and allowing them to self-feed by selecting and grasping food items themselves (Brown et al., 2017). Baby-led weaning has been associated with less picky eating (Brown & Lee, 2015; Taylor et al., 2017). Further evidence is needed to understand the impact of spoon-feeding versus a baby-led approach on child mealtime behavior.

This study is strengthened by using direct observations to examine parent-child feeding interactions during a typical dinner in the naturalistic home environment. However, one limitation is that observations could impact parents' usual feeding practices during mealtimes and this study was based on a single home observation. Parent feeding practices may vary depending on the food served during the meal and child behavior. Although families were instructed to perform their typical mealtime routine, we did not assess whether mealtimes were representative of participants' typical mealtime routine. Another limitation is that parent feeding

practices were coded for the mother and father if both were present at the mealtimes. Coding maternal and paternal feeding practices may provide a more nuanced picture of dyadic and whole-family mealtime interactions. Finally, picky eating was measured based on parent-self report, which is subject to response bias, including social desirability bias. Future studies should consider observing both parent feeding practices and child eating behavior to examine the bidirectional nature of parent-child feeding interaction.

## 6.5 Conclusion

Nonresponsive feeding practices were predictive of higher picky eating behavior, suggesting that responsive feeding practices may promote the development of healthful eating behavior in young children. Additional research is needed to better understand the relation between parental feeding responsiveness and child picky eating behavior.

## 6.6 Tables

**Table 6.1.** Characteristics of study participants (*n* = 91)

| | Mean ± SD or N (%) |
|---|---|
| **Parent characteristics** | |
| Age (years) | 31.0 ± 4.3 |
| Education level | |
| Grade school/high school | 1 (1.1) |
| Some college or technical school | 15 (16.5) |
| College graduate/post-graduate work | 72 (79.1) |
| Missing/unknown | 3 (3.3) |
| Employment status | |
| Employed | 64 (70.3) |
| Unemployed | 5 (5.5) |
| Stay-at-home parent | 18 (19.8) |
| Missing/unknown | 4 (4.4) |
| | |
| **Child characteristics** | |
| Age (months) | 21.2 ± 2.7 |
| Female | 46 (50.6) |
| Race | |
| Non-Hispanic/Latino White | 75 (82.4) |
| Non-Hispanic/Latino Non-White | 7 (7.7) |
| Hispanic/Latino | 3 (3.3) |
| Missing/unknown | 6 (6.6) |

**Table 6.2.** Means, standard deviations, and minimum and maximum values for observed parent feeding practices

| | Mean | SD | Range |
|---|---|---|---|
| Total feeding practices[a] | 40.14 | 21.29 | 5 to 102 |
| Responsive feeding practices[b] | 26.32 | 16.97 | 4 to 79 |
| Arrange food to make it interesting | 0.02 | 0.21 | 0 to 2 |
| Helps the child | 4.63 | 6.94 | 0 to 60 |
| Allows child to choose from available options | 5.66 | 5.16 | 0 to 21 |
| Asks questions about the food | 9.71 | 8.60 | 0 to 44 |
| Reasons with the child | 0.32 | 0.74 | 0 to 4 |
| Praises/compliments/approves/agrees | 1.81 | 2.41 | 0 to 13 |
| Positive comments about the food | 4.16 | 4.51 | 0 to 21 |
| Nonresponsive feeding practices[c] | 11.47 | 10.15 | 0 to 46 |
| Spoon feeds | 4.01 | 6.95 | 0 to 43 |
| Physically struggles | 0.37 | 1.13 | 0 to 8 |
| Suggests | 0.27 | 0.62 | 0 to 3 |
| Tells child to eat | 3.89 | 4.55 | 0 to 22 |
| Hurries child to eat | 0.00 | 0.00 | 0 |
| Begs child to eat | 0.08 | 0.31 | 0 to 2 |
| Promises food reward | 0.29 | 0.83 | 0 to 6 |
| Promises other reward | 0.10 | 0.58 | 0 to 5 |
| Threatens food punishment | 0.09 | 0.46 | 0 to 4 |
| Threatens other punishment | 0.02 | 0.15 | 0 to 1 |
| Disapproves/scolds | 1.96 | 3.23 | 0 to 16 |
| Negative comments about the food | 0.40 | 1.89 | 0 to 16 |

[a] Mean of all 19 items from the Feeding Behavior Coding System measured as frequency counts
[b] Mean of seven child-centered items from the Feeding Behavior Coding System measured as frequency counts
[c] Mean of 12 parent-centered items from the Feeding Behavior Coding System measured as frequency counts

**Table 6.3.** Bivariate correlations for reported and observed parent feeding practices[a]

| **Reported**[b] | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** |
|---|---|---|---|---|---|---|---|---|
| 1. Helps child | – | – | – | – | – | – | – | – |
| 2. Allows child to choose | .03 | – | – | – | – | – | – | – |
| 3. Asks questions | .17 | .11 | – | – | – | – | – | – |
| 4. Praises/approves | .17 | .04 | .28** | – | – | – | – | – |
| 5. Positive comments | .18 | .14 | .53*** | .52*** | – | – | – | – |
| 6. Spoon feeds | .12 | −.01 | −.26* | .19 | .02 | – | – | – |
| 7. Tells child to eat | .29** | .09 | .18 | .46*** | .45*** | .16 | – | – |
| 8. Disapproves/scolds | .05 | .12 | .02 | .23* | .19 | .22* | .38** | – |
| **Observed**[c] | | | | | | | | |
| 9. Helps child | −.04 | −.07 | .21 | .01 | .13 | .07 | −.06 | −.04 |
| 10. Allows child to choose | .04 | .19 | .17 | .13 | .24* | −.02 | .04 | −.04 |
| 11. Asks questions | .03 | −.00 | .17 | −.09 | .04 | −.19 | −.11 | −.18 |
| 12. Praises/approves | −.20 | −.28** | .06 | .21* | .22* | −.05 | .16 | −.01 |
| 13. Positive comments | −.06 | −.06 | .16 | .12 | .18 | .01 | −.02 | .05 |
| 14. Spoon feeds | .05 | −.03 | −.01 | .19 | .20 | .21* | .03 | .02 |
| 15. Tells child to eat | −.08 | −.14 | −.01 | .24* | .15 | .08 | .25* | .00 |
| 16. Disapproves/scolds | −.02 | −.11 | −.07 | .12 | −.00 | .11 | −.02 | −.17 |

* $p < .05$
** $p < .01$
*** $p < .001$

[a] Does not include feeding practices that occurred an average of less than .5 times per meal: arranging the food to make it more interesting for children, reasons with the child, physically struggles, suggests, hurries the child to eat, begs the child to eat, promise food reward, promising a reward other than food, threatens food punishment, threatens other punishment, makes negative comments about the food
[b] As measured by the Caregiver Feeding Styles Questionnaire (CFSQ)
[c] As measured by the Feeding Behavior Coding System (FBCS)

**Table 6.3. (cont.)**

| **Reported**[b] | **9** | **10** | **11** | **12** | **13** | **14** | **15** | **16** |
|---|---|---|---|---|---|---|---|---|
| 1. Helps child | – | – | – | – | – | – | – | – |
| 2. Allows child to choose | – | – | – | – | – | – | – | – |
| 3. Asks questions | – | – | – | – | – | – | – | – |
| 4. Praises/approves | – | – | – | – | – | – | – | – |
| 5. Positive comments | – | – | – | – | – | – | – | – |
| 6. Spoon feeds | – | – | – | – | – | – | – | – |
| 7. Tells child to eat | – | – | – | – | – | – | – | – |
| 8. Disapproves/scolds | – | – | – | – | – | – | – | – |
| **Observed**[c] | | | | | | | | |
| 9. Helps child | – | – | – | – | – | – | – | – |
| 10. Allows child to choose | .05 | – | – | – | – | – | – | – |
| 11. Asks questions | .31** | .35*** | – | – | – | – | – | – |
| 12. Praises/approves | .17 | .25* | .25* | – | – | – | – | – |
| 13. Positive comments | .20 | .24* | .37*** | .34*** | – | – | – | – |
| 14. Spoon feeds | −.08 | .25* | .02 | .28** | .18 | – | – | – |
| 15. Tells child to eat | .07 | −.07 | .22* | .39*** | .14 | .14 | – | – |
| 16. Disapproves/scolds | .11 | −.04 | .24* | .33** | .09 | .27** | .43*** | – |

* $p < .05$
** $p < .01$
*** $p < .001$

[a] Does not include feeding practices that occurred an average of less than .5 times per meal: arranging the food to make it more interesting for children, reasons with the child, physically struggles, suggests, hurries the child to eat, begs the child to eat, promise food reward, promising a reward other than food, threatens food punishment, threatens other punishment, makes negative comments about the food
[b] As measured by the Caregiver Feeding Styles Questionnaire (CFSQ)
[c] As measured by the Feeding Behavior Coding System (FBCS)

**Table 6.4.** Differences in observed parent feeding practices by reported feeding style

| | **Authoritative** | **Authoritarian** | **Indulgent** | **Uninvolved** | ***F* (3, 77)** |
|---|---|---|---|---|---|
| **Responsive feeding practices** | | | | | |
| Helps child | 1.18 | 1.70 | 1.49 | 1.06 | 1.73 |
| Allows child to choose | 1.48 | 1.59 | 1.50 | 1.10 | .84 |
| Asks questions | 1.80 | 2.35 | 2.18 | 1.95 | 1.54 |
| Praise/approves | .86 | .95 | 1.10 | .83 | .38 |
| Positive comments | 1.19 | 1.67 | 1.26 | 1.15 | 1.03 |
| **Nonresponsive feeding practices** | | | | | |
| Spoon feeds | 1.57 | 1.61 | .89 | 1.20 | 1.68 |
| Tells child to eat | $1.41_{a}$ | $1.72_{a}$ | $.88_{b}$ | $1.34_{ab}$ | 2.77* |
| Disapproves/scolds | .59 | .89 | 1.02 | 1.20 | 1.13 |

* $p < .05$

Note: means with different subscripts were significantly different from one another ($p < .05$). Feeding practices were natural log transformed.

**Table 6.5.** Regression analysis predicting children's food fussiness

| Variables | Model 1 | Model 2 | Model 3 | Model 4 |
|---|---|---|---|---|
| | β(SE) | β(SE) | β(SE) | β(SE) |
| Parent education level (ref: < high school) | .09(.19) | .10(.19( | .06(.17) | .05(.18) |
| Meal length (minutes) | −.17(.00) | −.16(.00) | −.18(.00) | −.17(.00) |
| Observed responsive feeding practices | | −.17(.02) | −.13(.02) | −.12(.02) |
| Observed nonresponsive feeding practices | | −.07(.03) | −.19(.02) | −.18(.02) |
| Reported responsive feeding practices | | | −.04(.15) | −.03(.16) |
| Reported nonresponsive feeding practices | | | **.46(.17)***** | **.50(.19)***** |
| Feeding style | | | | −.08(.09) |
| | | | | |
| $R^2$ | .01 | .02 | .19 | .18 |
| $F$ | 1.37 | 1.45 | **4.37***** | **3.78**** |
| $\Delta R^2$ | | .03 | **.14***** | .25 |
| $\Delta F$ | | 2.33 | **14.65***** | .42 |

* $p < .05$
** $p < .01$
*** $p < .001$

# CHAPTER 7:
# Cumulative Genetic Risk, Parent Feeding Responsiveness, and Child Picky Eating[7]


## Abstract

**Background:** Picky eating behavior is influenced by genetics and the environment. Previous gene-environment interaction studies have shown that children may be differentially affected by their rearing environment. Yet, it is unknown whether genes interact with parenting in the feeding context and if there is a joint influence on children's picky eating behavior.

**Objective:** The purpose of this study was to investigate the interaction of single nucleotide polymorphisms affecting dopamine signaling and parent feeding responsiveness on picky eating in young children.

**Methods:** Participants included 181 Caucasian children and their mothers who were participating in the STRONG Kids 2 longitudinal birth cohort. Mothers reported on their feeding practices and child's eating behavior when children were 24 months of age. Genomic DNA was obtained from saliva samples. Single nucleotide polymorphisms in dopaminergic genes were selected to form a cumulative genetic risk score (ranging from 0 to 6), which was then evaluated in moderating the effects of parent feeding responsiveness on child picky eating.

**Results:** A significant gene-environment interaction was observed. Contrary to our hypothesis, children with the highest cumulative genetic risk displayed more picky eating when exposed to an environment with high parent feeding responsiveness, and less picky eating in an environment with low parent feeding responsiveness.

**Conclusion:** Children vary in their response to parent feeding responsiveness dependent on their genetic make-up. This study contributes to understanding the moderating effects of genetics on the malleability of child eating behavior


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[7] To be submitted to *Current Developments in Nutrition*.

## 7.1 Introduction

Problematic eating behavior among children, such as picky or fussy eating, is a frequent cause of parent concern and mealtime stress (Harris et al., 2018; Rubio & Rigal, 2017; Trofholz et al., 2017). Although picky eating is defined many ways, the behavior is often characterized by having strong food preferences and consuming a narrow range of both new and familiar foods to the extent of interfering with the caregiver-child relationship (Lumeng, 2005; Taylor et al., 2015). Research shows that picky eating in children is related to disturbances in growth and development, including internalizing and externalizing behavioral problems (Cardona Cano et al., 2016; Hafstad et al., 2013b; Jacobi et al., 2008; Machado et al., 2016; Zucker et al., 2015). Additionally, studies suggest that picky eating and the acceptance of new foods is related to individual differences in child temperament (Bergmeier et al., 2014; Hafstad et al., 2013a; Jacobi et al., 2003; Moding et al., 2014; Pliner & Loewen, 1997).

It is well documented that genetic and environmental factors, including parent feeding practices, contribute to the development of children's eating behavior and food preferences (Birch & Fisher, 1998; Butte et al., 2006; Cooke et al., 2007; Fildes et al., 2014; Savage et al., 2007; Wardle & Cooke, 2008). According to the differential susceptibility hypothesis, individuals vary in their susceptibility to environmental influences based on genetics (Belsky et al., 2007; Belsky & Pluess, 2009). Taken together with the biological sensitivity to context hypothesis (Boyce & Ellis, 2005; Ellis & Boyce, 2008), children who are genetically susceptible to environmental influences (i.e., orchids) display more negative outcomes in unfavorable environments and more positive outcomes in favorable environments compared to children who are less genetically susceptible to environmental influences (i.e., dandelions). Previous gene-environment interaction (GxE) studies have shown that children may be differentially affected by

their rearing environment (Bakermans-Kranenburg & van Ijzendoorn, 2011; Belsky et al., 1998). However, to the best of our knowledge, no GxE study has investigated the rearing environment from the parent feeding context; thus, the joint influence of genetic and environmental factors on children's picky eating behavior is unknown.

The current study examines GxE involving the effects of cumulative genetic risk and parent feeding responsiveness on child picky eating behavior. Feeding responsiveness is the extent to which caregivers deliver prompt, contingent, and developmentally appropriate responses to the child's satiety and hunger cues, thus encouraging food acceptance (Black & Aboud, 2011; DiSantis et al., 2011; Pérez-Escamilla et al., 2017). A cumulative genetic risk score (Horne et al., 2005) was calculated based on genetic variants implicated in the dopaminergic system, which may influence child behavior by regulating the effectiveness of attention, motivation, learning, and reinforcement (Knafo et al., 2011; Papageorgiou & Ronald, 2013; Queiroz, 2004). Based on previous research identifying associations with child temperament or eating behavior, the following dopaminergic genes were selected for this study: dopamine receptor D4 gene (*DRD4*), dopamine receptor D2 gene (*DRD2*), catechol-*O*-methyltransferase (*COMT*), dopamine active transporter 1 gene (*DAT1*), and brain-derived neurotrophic factor gene (*BDNF*) (Davies et al., 2015; Laucht et al., 2012; Luijk et al., 2011; Obregon et al., 2017; Stice et al., 2010; Yeom et al., 2016). Consistent with differential susceptibility, we hypothesized that children with higher cumulative genetic risk (i.e., orchids) would display less picky eating behavior when exposed to an environment with high parent feeding responsiveness, but more picky eating behavior when exposed to an environment with low parent feeding responsiveness. The results in orchid children would be more pronounced compared to children with low cumulative genetic risk (i.e., dandelions).

## 7.2 Methods

### Participants

Mothers and infants were recruited as part of the Synergistic Theory Research Obesity and Nutrition Group (STRONG) Kids 2 birth cohort study in east-central Illinois. The STRONG Kids 2 (SK2) study aims to identify multi-level predictors of weight trajectories and dietary habits in children during the first five years of life, including factors related to individual biology and the family environment. Child saliva samples were collected for genotyping when the child was 6 weeks of age and parents completed a comprehensive panel survey when the child was 24 months. Children with missing data on child picky eating or parent feeding responsiveness were excluded for the current analyses. A subsample of non-Hispanic white children ($n$ = 181; 53% girls) for whom complete genetic were available were included in the study. Parents provided written consent for their child's participation in the study and were compensated for their participation. The University of Illinois Institutional Review Board approved this study.

### Measures

#### *Child Picky Eating Behavior*

The food fussiness subscale of the Children's Eating Behavior Questionnaire was used to measure parent's perceptions of their child's picky eating behavior (Wardle et al., 2001). Parents reported the frequency of their child's selectivity (e.g., "My child is difficult to please with meals") or pickiness regarding the range of accepted and rejected foods (e.g., "My child enjoys a wide variety of foods", reverse-coded). The food fussiness subscale contains six questions that were rated on a 5-point Likert scale (never to always). Higher mean food fussiness scores indicated higher picky eating behavior. In the present study, the food fussiness subscale showed high reliability (Cronbach's alpha = 0.90).

### *Parent Feeding Responsiveness*

The Caregiver Feeding Styles Questionnaire (CFSQ) was used to measure parent feeding responsiveness, which refers to the level of warmth and nurturance parents use in directing their children during meals (Hughes et al., 2005; Hughes et al., 2011). Parents reported their frequency of engaging in 19 feeding directives scored on a 5-point Likert scale (never to always). Feeding responsiveness is the mean of seven child-centered feeding directives divided by the mean of all 19 feeding directives. Child-centered feeding promotes child autonomy and self-regulation of intake (e.g., reasoning). Similar to previous work using a variable-centered approach (Morrison et al., 2013), we used a continuous score for feeding responsiveness.

### *Cumulative Genetic Risk*

Single nucleotide polymorphisms (SNPs) were genotyped using Fluidigm® Genotyping Analysis version 4.1.2 (San Francisco, CA, USA). Cumulative genetic risk was calculated based on genotyped polymorphisms using an additive model. Specifically, the A (Met) allele of *BDNF* rs6265, G allele (Val) of *COMT* rs4680, C allele of *DAT1* rs40184, T allele of *DRD2* rs1800497 and rs2283265, and the T allele of *DRD4* rs1800955 were identified as risk alleles. Each polymorphism was assigned one point for each risk allele present, then values were summed to create a cumulative risk score. The rs6265, rs4680, and rs1800955 SNPs were not consistent with Hardy-Weinberg equilibrium and were not included in the calculation of cumulative genetic risk. Therefore, polymorphisms in rs40184, rs1800497, and rs2283265 were summed for a total score ranging from 0 to 6. Based on the frequency of responses (Belsky & Beaver, 2011), cumulative risk scores were categorized as low (0 or 1 risk alleles), medium (2 or 3 risk alleles), and high (4, 5, or 6 risk alleles). The distribution of 0 or 1, 2 or 3, and 4-6 risk alleles was, respectively, 24.3% ($n$ = 44), 59.7% ($n$ = 108), and 16.0% ($n$ = 29).

**Statistical Analyses**

All statistical analyses were conducted using SAS version 9.4 (SAS Institute, Cary, NC, USA) and significance levels were set at $p < 0.05$. To distinguish between gene-environment correlation and gene-environment interaction, bivariate correlations were estimated to determine the association between cumulative genetic risk scores and parent feeding responsiveness. Generalized linear models were used to determine the main effects and multiplicative interaction of cumulative genetic risk and parent feeding responsiveness in predicting food fussiness scores. Post-hoc slope analysis was conducted on the interaction between cumulative genetic risk and parent feeding responsiveness to compare differences in picky eating behavior by cumulative genetic risk groups (i.e., low, medium, and high) and to confirm that the slope of the high genetic risk group is significantly steeper than the slope of the low-risk group.

**7.3 Results**

On average, parent feeding responsiveness was 1.29 (*SD* = 0.16, Range = 0.94 to 1.79) and food fussiness was 2.68 (*SD* = 0 .76, Range = 1.00 to 5.00). There was no association between child sex and parent feeding responsiveness or child food fussiness. There was no significant bivariate association between cumulative genetic risk scores and parent feeding responsiveness ($r$ = -.09, $p$ = 0.24), indicating that any GxE was not a simple reflection of gene-environment correlation.

A significant interaction of cumulative genetic risk and parent feeding responsiveness was found for food fussiness, $F(2, 175) = 3.64$, $p = 0.03$. There was a significant main effect for cumulative genetic risk, $F(2, 175) = 3.42$, $p = 0.03$ but not for parent feeding responsiveness, $F(1, 175) = 3.54$, $p = 0.06$. **Table 7.1** displays the simple slopes for each of the cumulative

genetic risk groups. Slopes for parent feeding responsiveness increased in magnitude when moving from the low-risk score group (i.e., 0 or 1 risk alleles) to the high-risk score group (i.e., 4, 5, or 6 risk alleles). The slope for the high-risk score group was significantly different from the slopes for the low- or medium-risk score group ($t = 2.70$, $p < 0.05$ and $t = 2.33$, $p < 0.05$, respectively). There was no significant difference in slopes between the low- and medium-risk score group. Slopes for each cumulative risk group were plotted across values of parent feeding responsiveness (**Fig. 7.1**). Respondents with 4, 5, or 6 risk alleles had the highest food fussiness scores when exposed to the environment with high feeding responsiveness and the lowest food fussiness scores when exposed to the environment with low feeding responsiveness. The reverse pattern was detected for respondents with 0 or 1 risk alleles and 2 or 3 risk alleles, as they scored the lowest on food fussiness in an environment with high feeding responsiveness.

### 7.4 Discussion

Our results suggest that children, for reasons based on genetic differences, vary in the extent to which their picky eating behavior is apparently affected by parent feeding responsiveness. To the best of our knowledge, this is the first study to report a significant Gene x Parenting interaction predicting child picky eating behavior. Carrying more risk alleles amplified the effects of parent feeding responsiveness on picky eating. However, contrary to the hypothesis, the interaction was not aligned with the theory of differential susceptibility in which individuals vary in their susceptibility to both positive and negative environmental influences "for better *and* for worse" (Belsky et al., 2007; Belsky & Pluess, 2009). Children with the highest genetic risk (i.e., the orchids) displayed *more* picky eating when exposed to an environment with high parent feeding responsiveness, and less picky eating in an environment

with low parent feeding responsiveness. Additionally, children with low genetic risk (i.e., the dandelions) were less picky when parents had high feeding responsiveness.

Parent feeding practices are a well-studied source of influence on children's picky eating behavior (Dovey et al., 2008). Children whose parents use non-responsive feeding practices, such as restricting food intake or pressuring, are more likely to be picky eaters (Galloway et al., 2005; Powell et al., 2011; van der Horst, 2012; Webber et al., 2010). Whereas children whose parents use responsive feeding practices, such as positive statements and family mealtime routines, tend to have better eating behavior, including more food enjoyment and less picky eating (Finnane et al., 2017; Steinsbekk et al., 2017). High parent feeding responsiveness reflects reciprocity between the parent and the child, and if parents lack responsivity during mealtimes then children may resort to picky eating behavior (Black & Aboud, 2011). One potential reason why orchid children were picky eaters with high parent feeding responsiveness could be that they are less sensitive to environmental influences, such as the reward or reinforcement value of their parents' feeding interactions. Previous work on differential susceptibility suggests that orchid children may need more emphasis on the reward value of parent's responses to their actions (Bakermans-Kranenburg et al., 2008). The use of extrinsic rewards is a common strategy used by parents to encourage food consumption (Orrell-Valente et al., 2007). However, offering tangible rewards for eating, particularly food rewards, may have negative effects on children's eating behavior (Cooke et al., 2011; Roberts et al., 2018). Parents of orchid children may need to make mealtimes more intrinsically rewarding, such as verbalizing praise during mealtime and establishing pleasant and structured family meals. Another possibility why our findings were opposite to the hypothesis is that the benefits of parent feeding responsiveness on child picky eating behavior may be more evident in the long-run when children are older.

Important limitations to this study are that parent feeding responsiveness and child picky eating were assessed through self-report, which could potentially bias information about parent-child mealtime interactions. Observational measures of parent and child mealtime behavior may capture data on feeding interactions that may not be evident from self-reported measures (Pesch & Lumeng, 2017). This study is also limited in that the approach assumes the selected polymorphisms act additively to influence dopamine signaling. Genetic risk scores were not weighted according to predicted effect size and potential multiplicative relations were not considered. Furthermore, other alleles commonly implicated in the dopaminergic system were not included (e.g., the *DRD4* 7-repeat allele, *DAT1* 10-repeat allele, *MAOA* 2R/3R alleles, and the 5HTTLPR short allele). Future studies should consider collecting molecular data from both parents and children as shared genes could influence parent behavior. Finally, although a significant cross-over interaction was observed, the relation was not consistent with differential susceptibility. This study only assessed parental feeding responsiveness (a positive environmental influence), and better measurement of the feeding environment is key in identifying the interplay between genetics and parenting on child eating behavior. Future studies should consider measuring children's susceptibility to negative parental feeding influences (e.g., feeding non-responsiveness) as this may have a greater impact on picky eating behavior compared to supportive parental feeding influences. Evaluating both positive and negative feeding environmental influences may inform interventions that not only reduce the presence of adverse parental feeding behavior, but also promote supportive parental feeding behavior. Additional work is also needed to investigate the interaction between genetics and parental feeding responsiveness on other child eating behavior outcomes, such as fruit and vegetable consumption.

## 7.5 Conclusion

Children vary in their response to parent feeding responsiveness dependent on their genetic make-up. Specifically, the association between parent feeding responsiveness and picky eating behavior is accentuated under conditions of higher cumulative genetic risk. However, contrary to the theory of differential susceptibility, children with the highest genetic risk displayed more negative eating behavior when exposed to a supportive environment. This study contributes to understanding the moderating effects of genetics on the malleability of child eating behavior. Additional research is needed to understand the etiology of picky eating behavior in children, and how parent feeding responsiveness interacts with other genetic factors in shaping children's eating behaviors.

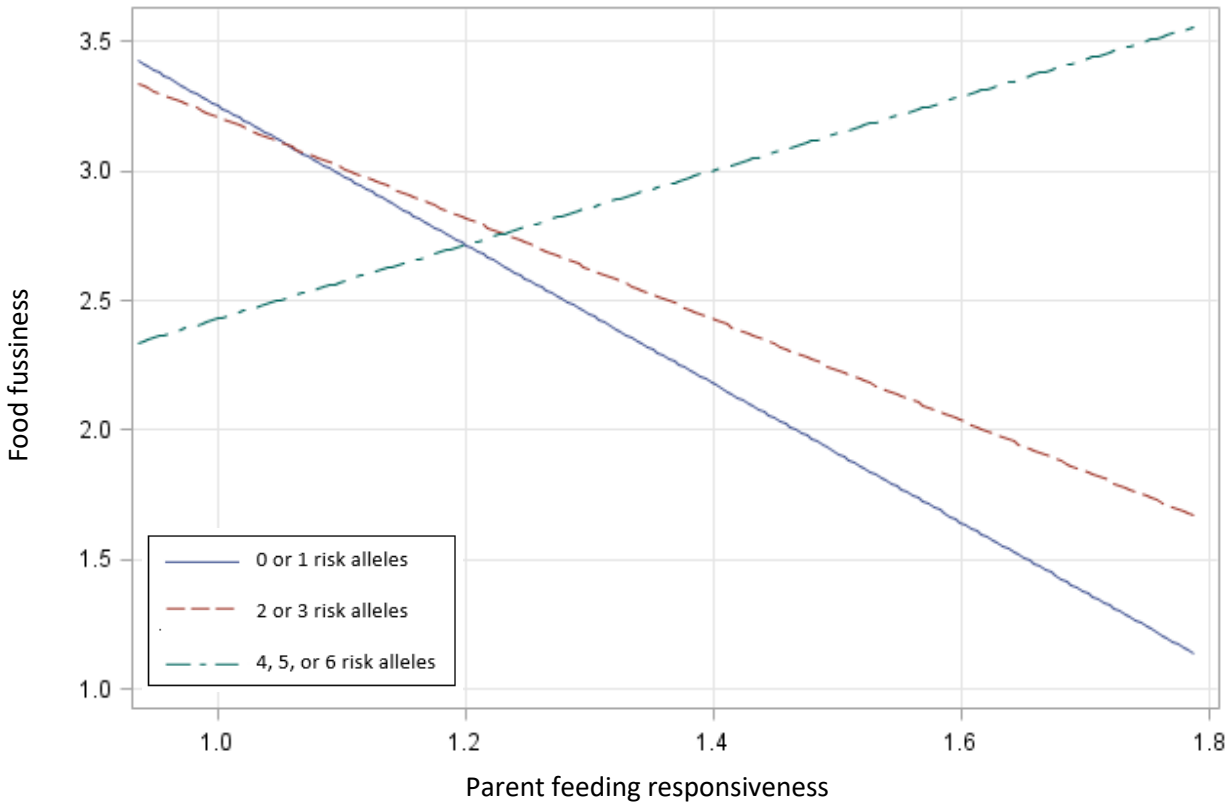
## 7.6 Tables and Figures

**Table 7.1.** Post-hoc analysis of the gene-environment interaction between cumulative genetic risk and parent feeding responsiveness

| | Simple Slope | Comparison group for t-test for equality in regression slopes | |
|---|---|---|---|
| | Parent Feeding Responsiveness | Low risk score (0 or 1 risk alleles) | Medium risk score (2 or 3 risk alleles) |
| Low risk score (0 or 1 risk alleles) | -2.68 | - | - |
| Medium risk score (2 or 3 risk alleles) | -1.95 | 0.87 | - |
| High risk score (4, 5, or 6 risk alleles) | 1.43 | 2.70* | 2.33* |

* $p < 0.05$, two-tailed tests

**Figure 7.1.** Interaction between cumulative genetic risk and parent feeding responsiveness in the prediction of picky eating



# CHAPTER 8:
# Conclusions

## 8.1 Summary

This dissertation undertook a transdisciplinary approach to understanding picky eating behavior in young children. The overall objective of the studies presented herein was to determine the influence of genetics (nature) and the feeding environment (nurture) on picky eating behavior in young children. Prior to this research it was understood that children's eating behavior is influenced by both genetic and environmental factors, including parent feeding practices. However, no study had investigated the joint influence of nature and nurture on children's picky eating behavior using a gene-environment (GxE) interaction approach.

To investigate the role of nature on picky eating, we conducted two studies investigating the association between candidate genetic variants and picky eating behavior. Parents completed questionnaires about the frequency of their child's picky eating behavior, and the child's saliva was collected for genotyping of single nucleotide polymorphisms (SNPs). In support of our hypothesis, genes related to chemosensory perception (Chapter 3) and appetite control (Chapter 5) were associated with picky eating behavior in children. Our findings are consistent with previous research indicating that picky eating has a strong genetic component. We found that variation in genes related to bitter taste was associated with picky eating, suggesting that children who are sensitive to bitter taste are more likely to be picky eaters. We also discovered that variation in genes relate to appetite regulation are associated with picky eating, suggesting that picky eaters may have reduced appetite based on genetic differences.

The next step was to investigate the role of nurture, in which we conducted two studies to examine the influence of the feeding environment on picky eating behavior. In a prospective study, we found that several factors in the home feeding environment predicted picky eating in

preschool-aged children one year later (Chapter 4). Children's picky eating behavior was improved by avoiding the television, maintaining parent control of food choices during mealtimes, and having a higher sense of positive climate during family meals. In the second study, we observed families during dinner in the naturalistic home setting and analyzed video-recordings of parent feeding practices (Chapter 6). Consistent with our hypothesis, we found that parental responsive feeding practices (i.e., practices that foster child intake self-regulation and recognition of internal satiety and hunger cues by exhibiting warmth and attunement around meals) were inversely associated with picky eating in young children.

Finally, a GxE interaction approach was used to investigate the joint influence of nature and nurture on picky eating (Chapter 7). A cumulative genetic risk score was calculated based on genetic variants (SNPs) previously implicated in the dopaminergic system. Based on the concept of differential susceptibility to the rearing environment, we hypothesized that children with higher cumulative genetic risk would display less picky eating when exposed to an environment with high parental feeding responsiveness, but more frequent picky eating in an environment with low parental feeding responsiveness. We found a significant GxE interaction, but the relation was contrary to our hypothesis. Children with the highest cumulative genetic risk were pickier with high feeding responsiveness, and less picky with low feeding responsiveness.

Are picky eaters born or made? Collectively, these results indicate that both nature and nurture influence the development of picky eating in children. Furthermore, children vary in their response to the feeding environment dependent on their genetic make-up. Overall, these findings can be used to inform the development of anticipatory guidance for parents and caregivers of young children. Additional research is needed to better understand the etiology of picky eating behavior in young children.

## 8.2 Future Directions

This research identified specific genetic and environmental factors related to picky eating in young children. However, there are several recommendations for future research to build on these findings.

First, picky eating represents one aspect of child eating behavior. Research needs to be expanded to broader categories of child eating behavior, particularly other "food avoidance" subscales of the Child Eating Behavior Questionnaire (CEBQ), such as satiety responsiveness, emotional undereating, and slowness in eating (Sleddens et al., 2008; van der Horst & Sleddens, 2017; Wardle et al., 2001). Assessing other food avoidant subscales could identify eating behaviors that could be implicated in feeding problems and risk of underweight (Powell et al., 2011). Measuring food avoidance could also identify other problematic mealtime behaviors that are not captured by picky eating questionnaires. Future studies should also delineate between picky eating and food neophobia, which are related constructs with a common etiology, but are theoretically different (Dovey et al., 2008; Smith et al., 2017).

Second, although this research included behavioral mealtime observations, picky eating was measured based only on parental perception. Self-reported measures of picky eating are a subjective assessment of child eating behavior and can vary among caregivers (Luchini et al., 2017a). Parents may not be able to accurately estimate how much their child's picky eating deviates from typical eating behavior in children (Werthmann et al., 2015). Observational measures of picky eating could identify unique aspects of child eating behavior and the bidirectional feeding relationship that may not be captured by parental report (Edelson et al., 2016; Fries et al., 2017; Luchini et al., 2017b; Walton et al., 2017). Additionally, behavioral coding of mealtime observations can provide rich qualitative and quantitative data (Pesch &

Lumeng, 2017). Future work should consider the use of both reported and observational measures of picky eating to assess correlations and measure multiple constructs of the behavior.

Third, longitudinal studies are needed to elucidate the course of picky eating in toddlers as well as the stability of the behavior between early childhood, adolescence, and adulthood. Participants in this study were part of the Synergistic Theory Research Obesity and Nutrition Group (STRONG) Kids 2 (SK2) birth cohort, which follows participants over the first five years of the child's life. Questions related to picky eating were included in the panel survey at 12, 18, 24, 36, 48, and 60 months. Thus, future research specific to the SK2 birth cohort would be to investigate the trajectory of picky eating from age 1 to 5 years. Furthermore, the SK2 survey includes two other questionnaires on problematic eating behavior in addition to the CEBQ food fussiness subscale that can be used to develop a more comprehensive measure of picky eating – the Mealtime Assessment Survey (Boquin et al., 2014b) and the food refusal subscale from the Oregon Research Institute Child Eating Behavior Inventory (Lewinsohn et al., 2005).

Finally, while this research did involve the measurement of cumulative genetic risk using genes implicated in the dopaminergic system, it did not include several genetic polymorphisms that are commonly examined in GxE interaction studies involving the differential effects of parenting on child behavioral outcomes. Other genetic markers related to differential susceptibility to the environment include the 7-repeat allele of the dopamine receptor D4 (*DRD4*) gene, the 10-repeat allele of the dopamine transporter 1 (*DAT1*) gene, the low-activity allele of the monoamine oxidase A (*MAOA*) gene, the T allele of the serotonin receptor gene (*HTR2A*), and the short allele of the serotonin-transporter-linked polymorphic region (*5-HTTLPR*) in the *SLC6A4* gene (Belsky & Pluess, 2009). Extending the genetic component of the GxE interaction is an important step in gaining a better understanding of the etiology of picky eating.

## CHAPTER 9:
## References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## APPENDIX A:
## DRD4 Genotyping Method

1. Primer Design

| Region | Dye | Primer Name | Primer DNA sequence | Protocol | ug DNA/rxn | Ta | Sizes |
|---|---|---|---|---|---|---|---|
| DRD4 Ex3 | FAM | DRD4-3 | 5'-FAM-GCG ACT ACG TGG TCT ACT CG-3' | DYN | 50 | 55 | 318-364-410-428-458-502-546-594 |
| | | DRD4-R1 | 5'-(GTTTCTT)GGT CTG CGG TGG AGT CTG-3' | | | | |

2. PCR Reaction

| **Master Mix (Anchordoquy modification)** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Component** | **Per Rxn (µL)** | **2.5** | **5** | **8** | **10** | **100** | **110** | **150** |
| $dH_2O$ | **7.4** | 18.5 | 37.0 | 59.2 | 74.0 | 740.0 | 814.0 | 1110.0 |
| $MgCl_2$ (25mM) | **1.6** | 4.0 | 8.0 | 12.8 | 16.0 | 160.0 | 176.0 | 240.0 |
| DMSO | **2** | 5.0 | 10.0 | 16.0 | 20.0 | 200.0 | 220.0 | 300.0 |
| F Primer (10µM) | **0.5** | 1.3 | 2.5 | 4.0 | 5.0 | 50.0 | 55.0 | 75.0 |
| R Primer (10µM) | **0.5** | 1.3 | 2.5 | 4.0 | 5.0 | 50.0 | 55.0 | 75.0 |
| dNTPs | **0.8** | 2.0 | 4.0 | 6.4 | 8.0 | 80.0 | 88.0 | 120.0 |
| Buffer (10X) | **2** | 5.0 | 10.0 | 16.0 | 20.0 | 200.0 | 220.0 | 300.0 |
| Taq (5U/µL) | **0.2** | 0.5 | 1.0 | 1.6 | 2.0 | 20.0 | 22.0 | 30.0 |
| Total Volume (µL) | **15** | 37.5 | 75.0 | 120.0 | 150.0 | 1500.0 | 1650.0 | 2250.0 |

| **DRD4 PCR Setup** | |
|---|---|
| Mastermix | 15 µL |
| DNA | 5 µL |
| Total Volume | 20 µL |

| | | | | |
|---|---|---|---|---|
| | 1 | cycle of | 95.0°C | 10 min |
| | 40 | cycles of | 95.0°C | 30 sec |
| | | | 65.0°C | 30 sec |
| | | | 72.0°C | 3 min |
| | 1 | cycle of | 72.0°C | 10 min |
| | 1 | hold at | 4.0°C | forever |

**APPENDIX B:**
**Parent Feeding Responsiveness Codebook**

These codes capture specific feeding behaviors exhibited by the parent and the frequency of occurrence of these behaviors during the mealtime.

## Procedures & Definitions

**Frequency coding:** This coding scheme involves the number of times the parent administers a particular feeding behavior during the mealtime. Each parent will be scored on the number of times they engage in a particular feeding behavior from start of mealtime to end of mealtime.

**Mealtime start definition**: The start of the mealtime is defined by the time at which food is placed in front of the target child. If food is already in front of child when the camera is on, the start of the mealtime is defined by the time at which the camera is turned on.

**Mealtime end definition**: The end of the mealtime is defined by the time at which food is taken away from the target child, or at 20 minutes—whichever occurs first.

**Feeding Responsiveness:** number of child-centered feeding behaviors engaged by the parent divided by the total number of feeding behaviors engaged by the parent. Child-centered feeding behaviors promote child autonomy (behaviors #1, 3, 4, 7, 12, 19, 21)

## Feeding Behavior Coding

- **Situational Management**

1. **Arranging the food**
   Parent makes the food look more interesting in order to entice the child into eating
   *Examples:*
   - Making a sandwich, celery stick, and raisins to look like a butterfly.
   - Putting smiley faces on pancakes.
   - Making boats of cucumber and tuna.

2. **Serves**
   Parent serves the child's food – this may also denote the start of mealtime

3. **Helps**
   Parent verbally or physically enables the child to eat other than spoon feeding → be sure to differentiate from 'Physically intervenes'.
   *Examples:*
   - Gives verbal instructions on serving self.
   - Gives hands-on training on serving, scooping food into spoon or fork.
   - Cuts up food into bite-sized pieces to make more manageable.
   - Parent brings plate or utensils closer to the child.

4. **Allows the child to choose**
   Parent allows the child to decide what he/she would like to eat from appropriate options.
   *Examples:*
   - "We have corn, beans, and meat. What would you like to eat?"
   - Parent offers condiments to accompany meal (e.g., butter, mustard, ketchup)
   - Child: "I don't like it." Mom: "That's fine; you don't have to eat it."

5. **Spoon feeds**
   Parent gives the child food (e.g., via utensil or directly) in order to get the child to eat
   *Example:*
   - Parent picks up hot dog and places it in front of child's mouth.

6. **Physically intervenes**
   Parent touches, grabs, or places the child in order to get him or her to eat → be sure to differentiate from 'Helps'.
   *Examples:*
   - Parent grabs child by arm while in front of the plate directing child to eat.
   - Parent picks up the child and places him or her into their seat to eat.
   - Brings chair/child closer to table to get child to eat.

- **<u>Verbal Directives</u>**

7. **Asks questions**
   Parent asks child questions <u>about the food</u> whether the child is eating or not
   *Examples:*
   - "What is the name of that food?"
   - "What color are the peas?"
   - "How many pieces of meat do you have left?"
   - "Doesn't this look good?"

8. **Verbally offers child 2nd helping**
   Parent <u>verbally</u> offers 2nd helping of food to child –may occur with permissive behavior
   *Example:*
   - "We have corn, beans, and meat. Would you like a second helping?"

9. **Suggests**
   Parent subtly implies that they want the child to eat
   *Example:*
   - "Bobby, your dinner is getting cold."

10. **Tells**
    Parent verbally directs child to eat meal or to eat a specific item on the plate
    *Examples:*
    - "Eat your dinner."
    - "Eat a little bit of your carrots."
    - "Eat all of your vegetables."

**11. Hurries**

Parent rushes child to eat

*Example:*

- "Hurry up and eat your food."

**12. Reasons**

Parent engages the child to eat the food using reasons or rationales

*Examples:*

- "Meat has lots of iron."
- "Milk makes strong bones."
- "Carrots are good for your eyes."
- "Putting the sauce on top makes it taste better."

**13. Begs the child to eat**

Parent pleads with the child in order to get him to eat

*Example*:

- "Please, please, please eat your soup."

**14. Comparison**

Parent compares the child's eating to another person in the family.

*Examples*:

- "Look how good your brother is at eating his vegetables"
- "Why don't you eat your carrots like your sister?"

- **<u>Threat/Bribe</u>**

**15. Threatens food punishment**

Parent tells child there will be consequences regarding food if he/she doesn't eat.

*Examples*:

- "If you don't eat the rest of your meat, you can't have any more potatoes."
- "You guys are not getting anything else to eat until you finish your dinner."

**16. Promises food reward**

Parent tells the child that he/she will get a food reward for eating (any amount).

*Example*:

- "If you eat the rest of your meat, you can have dessert."

**17. Threatens other punishment**

Parent tells child they will take away something other than food if he/she does not eat.

*Example*:

- "If you don't eat your food, you won't get to go outside and play."

**18. Promises other reward**

Parent tells the child that he/she will get a reward (not food) for eating (any amount).

*Examples***:**

- "If you eat your food, you can go outside and play when you are finished."
- "If you eat your food, I will give you a sticker when you are finished.

- **Scold/Praise**

**19. Praises/compliments/approves/agrees (related to mealtime behavior)**

Parent praises the child for eating (this category includes approval and agreement)

*Examples*:

- "You are such a good boy for eating your lunch!"
- "You did a good job of eating your peas!"
- "Good idea mixing the two foods together on your plate!"

**20. Disapproves/scolds (related to mealtime behavior)**

Parent shows verbal disapproval for the child not eating/mealtime behavior

*Examples***:**

- "You know I don't like it when you don't eat your food."
- "Quit eating so much bread."
- "You haven't even eaten your vegetables."
- "Stop banging your spoon on your plate."

**21. Positive comment/enthusiastic modeling (related to food)**

Parent says something positive about the food the child is eating or was served in order to get him or her to eat

*Examples*:

- "Those potatoes you are eating really look good!"
- "Mmm these are tasty."

**22. Negative comments (related to food)**

Parent says something negative about the food the child is eating in order to discourage overeating or to discourage choice of food

*Examples:*

- "Those French fries and ketchup don't look so good."
- "All of that ice cream is going to give you a headache."
- "Eating too much gravy will make you fat."
- "Mixing beans with mashed potatoes looks gross."

- **Permissive**

**23. Ignores child or shows indifference to child → Uninvolved**

Parent ignores the child, shows indifference to the child, or does not respond appropriately to the child's comments about the food or mealtime.

*Examples:*

- The child is talking about milk and parent does not respond to child's verbalizations
- The child is talking about the peas and carrots. The parent repeats "Peas and carrots" but does nothing to reinforce that peas and carrots are good for the child.

**24. Gives child multiple helpings/servings of food → Indulgent**
Parent indulges child by continually giving food with no regard to what is on the plate

**25. Allows child to take 2nd or 3rd helping → Indulgent**
Parent allows child to continue to take multiple servings of various foods.